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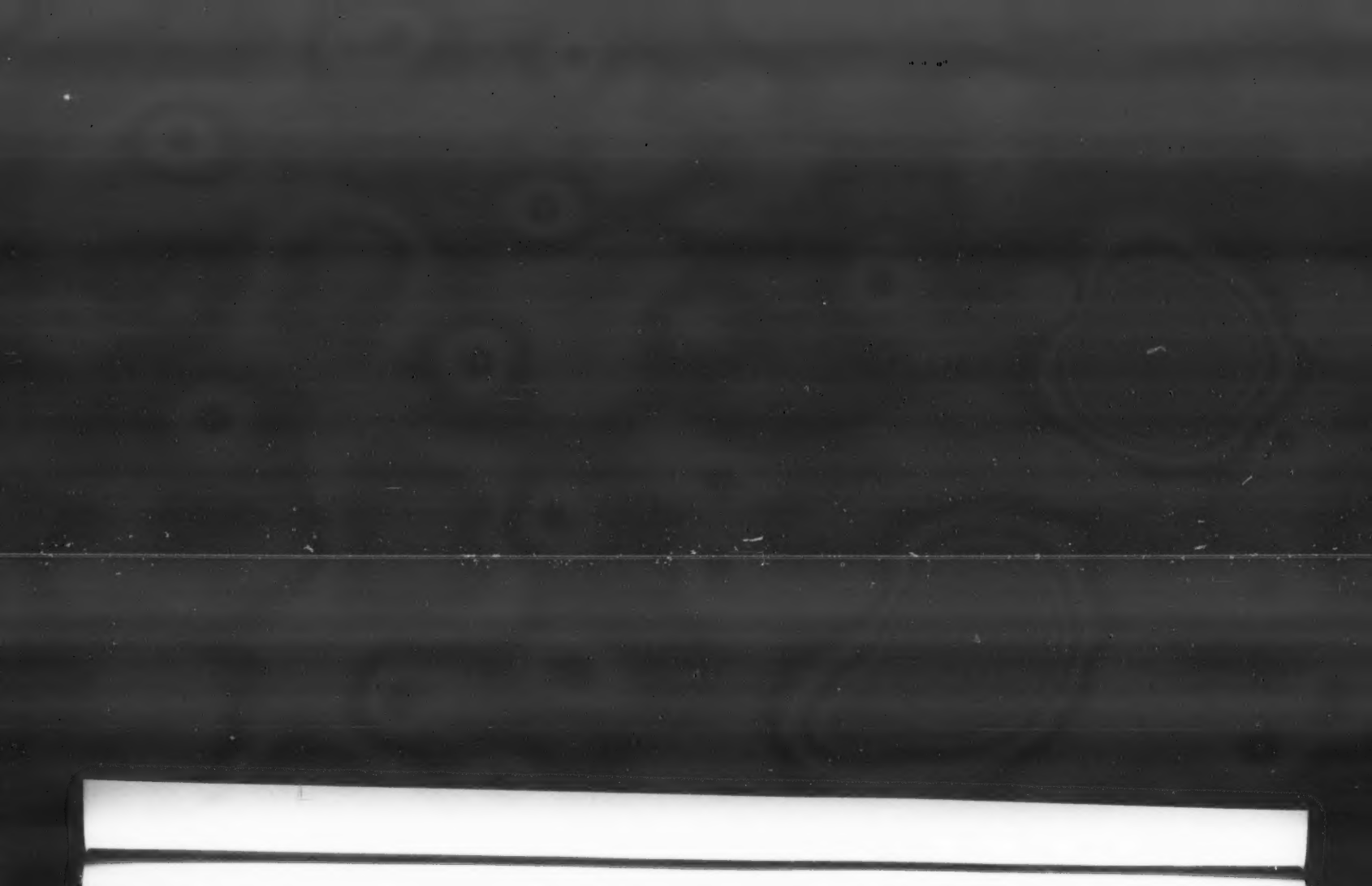

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JOHN HERR MUSSER, M.D., LL.D.

Not only the medical profession of Philadelphia, but also physicians throughout the United States, as well as his countless friends among medical men of other lands, were shocked by the sudden death of Dr. John Herr Musser, which occurred in Philadelphia on April 3, 1912, after a brief illness.

Dr. Musser was born in Strasburg, Lancaster County, Pennsylvania, June 22, 1856. It is interesting to note that Dr. Musser was descended from a remarkable line of physicians. Since the

early days of the colony of Pennsylvania, when William Penn granted to one of Dr. Musser's ancestors the right to practise, almost every generation of his family has contributed a representative to the medical profession: his great-grandfather, grandfather, and father all having been physicians of note in their respective communities.

After receiving his preliminary education at the Strasburg High School and Millersville State Normal School, Dr. Musser entered the Medical Department of the University of Pennsylvania, from which institution he received his medical degree in 1877. The following year he spent in the Philadelphia Hospital as a resident physician. Shortly after leaving the hospital he became officially connected with the University of Pennsylvania, which institution he was destined to serve continuously, with extraordinary loyalty and enthusiasm, until the time of his death.

No phase of Dr. Musser's career brought him more pleasure and satisfaction, as well as distinction, than his work as a teacher of medicine. He began as quiz master in the practice of medicine when that form of instruction occupied a well-recognized place in medical teaching. In 1881 he became instructor of clinical medicine in the University of Pennsylvania; eight years later he was appointed assistant professor of clinical medicine, and in 1898 he became professor of clinical medicine in that institution, a position which he filled with distinction until his death. Dr. Musser was essentially a clinical teacher. His keen power of observation, wide knowledge of medicine, and thorough understanding of pathological processes, coupled with a vast clinical experience, rendered him particularly well fitted for bedside instruction, in which he excelled. The thoroughness and painstaking detail with which he studied his patients, together with the energy and enthusiasm which marked his teaching, profoundly impressed his students. Although always alert to be among the first to adopt and advocate every advance in scientific medicine, he was ever mindful of the importance of teaching the art of medicine, and his clinics were replete with helpful and practical suggestions, long remembered by his hearers.

In addition to his activity as a teacher in the University of Pennsylvania, he labored unceasingly in numerous Philadelphia hospitals. In 1884, after serving as a dispensary physician, he became pathologist to the Presbyterian Hospital, and three years later was appointed a physician to that institution, a position

which he held at the time of his death. He served in the hospital of the University of Pennsylvania continuously from 1879, first as medical registrar, a number of years as chief of the medical dispensary, and, finally, as visiting physician. From 1885 he was connected for twenty years with the Philadelphia General Hospital as visiting physician, resigning to become consultant to that institution. Dr. Musser's deep interest in the better development of hospitals in this country claimed his active support in aiding and improving the organization and equipment of the institutions with which he was associated.

From the earliest days of his career until his death, Dr. Musser was a staunch supporter of everything that made for scientific medicine and the betterment of the medical profession. Although a clinician himself, he was keenly alive to the value of experimental medicine, and was virtually the founder of the Department of Research Medicine in the University of Pennsylvania. The Social Service Department now connected with the hospital of that institution owes its successful development largely to his energy and foresight. Always a firm believer in the closer association of physicians with each other, he was a prominent and leading figure not only in the various medical societies of his own city and State, but also in national organizations. The long list of distinguished positions held by him in various societies from time to time, testifies to the high regard in which he was held by the profession, particularly the signal honors which were conferred upon him by his election as president of the American Medical Association in 1904 and his appointment as chairman of the American Committee of the International Medical Congress at Budapest in 1909.

The unflagging energy and extraordinary capacity for work displayed by Dr. Musser are further emphasized by the number and importance of the contributions to medical literature which he found time to publish, notwithstanding the exacting claims of the active practice and multitudinous other interests in which he was engaged. His most notable contribution was his comprehensive work on *Medical Diagnosis*. In addition, he edited, in conjunction with the late Dr. A. O. J. Kelly, *Practical Treatment*, and was an extensive contributor to Keating's *Diseases of Children*, Hare's *System of Practical Therapeutics*, Nothnagel's *System of Medicine*, and Osler's *Modern Medicine*. Moreover, from 1884 until just before his death, Dr. Musser was a frequent

contributor to the various medical journals, and demonstrated his versatility by the ability and authority with which he wrote upon all phases of internal medicine.

Not only was he recognized as a physician of exceptional ability, but he was also greatly esteemed as a broad-minded, public-spirited citizen, eager to aid in every effort for civic betterment, and as a leader in every movement directed toward moral, hygienic, and social uplift. His kindly nature, wholesome optimism, and rugged honesty inspired confidence and affection in all who came under his influence.

Although his untimely death falls most heavily upon the community in which for thirty-five years he labored so untiringly, his loss will be felt by laymen as well as by the medical profession throughout the entire country. Dr. Musser set an example of unselfish, tireless devotion to duty and high professional ideals, supported by unfailing cheerfulness and courage, which can never be forgotten, and which will be a lasting inspiration to all who knew him. To those whose privilege it was to be closely associated with him, his memory will endure as the symbol of all that is best in professional attainment and truest in manhood.

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THE

AMERICAN JOURNAL

OF THE MEDICAL SCIENCES

MAY, 1912

ORIGINAL ARTICLES

PNEUMONIA.[1]

By J. T. Fotheringham, M.B.,

ASSOCIATE PROFESSOR OF MEDICINE AND CLINICAL MEDICINE IN THE UNIVERSITY OF TORONTO.

As the seasonal incidence of pneumonia has been distinctly in evidence in our work of the last three or four months, I have selected that familiar, not to say threadbare topic, not in the hope of writing anything particularly new, but in the desire to focus my experience and compare it with that of others in regard to certain phases, particularly of the etiology, clinical course, and prognosis, and especially the treatment.

Space will not permit of any reference to the history of this disease and its literature. "Lung fever" was well known to Hippocrates, and probably earlier, though pleurisy and pneumonia were not distinguished until Laennec, in 1819, described their morbid anatomy, and his work, with that of Auenbrugger, determined the physical signs by which diagnosis is now fairly easy and certain, particularly in the adult. Only so recently as the early eighties was the immediate cause finally determined. Klebs was the pathfinder in the bacteriology of the disease, as in 1877 he described a round motile micrococcus present in the lungs, sputum, and blood. In 1881 Eberth described a similar but different micrococcus, and in 1882 Koch described still another oval-shaped one. In the same year Friedländer detected the microbe named after him, and cultivated and inoculated it, producing a pneumonia which was patchy, but not lobar. Two years later, in 1884, during which time Friedländer's observations were held to be conclusive, Fraenkel

[1] Read at the March meeting of the Medico-Chirurgical Society, Ottawa.

and Weichselbaum separated and grew, and successfully inoculated, another microbe, the Diplococcus pneumoniæ, which has been proved to be much more frequent in pneumonia than Friedländer's microörganism; so that while Friedländer's bacillus is an important microbe of pneumonia, Fraenkel's diplococcus is the recognized cause of the great majority of cases of true lobar pneumonia. It has been clearly shown that the pneumococcus does not necessarily produce only lobar pneumonia, but may cause lobular lesions especially in children; also, that either of these forms of pneumonia may be produced by various organisms, such as the streptococcus, the staphylococcus, the bacilli of putrefaction, anthrax, plague, tubercle, typhoid, influenza, and others. It should be remembered that in pneumonia complicating, for example, typhoid, the lung infection is more apt to be due to the pneumococcus than to the organism causing the original disease. Also, that in some diseases, as influenza, the lung inflammation is apt to be due to the influenza bacillus, if it occur before the height of the illness and to the pneumococcus if the lung complication be a sequel. If it were possible to abandon the useful clinical and anatomical classification of lobar and lobular or bronchopneumonia in favor of the more scientific classification by the germ causing the disease, it might be well, but at present it is neither possible nor, from the point of view of treatment, necessary.

Without following the bacteriology of the disease further, one may define it in two senses—the broad and the narrow. In its broadest sense, acute pneumonia or pneumonitis is any inflammation of the lung, no matter how distributed anatomically, accompanied by acute inflammatory consolidation of the vesicular tissue. In its narrow sense the term is reserved for those consolidations, which form a single mass of considerable extent, generally spoken of as lobar. In the other form, originally designated as bronchopneumonia or lobular, the consolidations are numerous, small, and disseminated, involving, usually, the terminal bronchi primarily, and later their associated vesicles, or groups of vesicles.

Incidence. There is no disease more widespread in all parts of the earth and among all races. It is also one of the most fatal, as well as frequent, of acute diseases. The figures given are: 3 per cent. of all diseases and 6 per cent. of all medical diseases, while the mortality is 6.6 per cent. of all deaths and 12.7 per cent. of all deaths from medical diseases. The average mortality is not less than 1.5 to 2.3 per thousand persons living. I am not, I think, wronging the city of Chicago when I give from memory a statement made years ago that there the deaths anually from pneumonia outnumbered those from tuberculosis. The United States Census of 1900 gave 111,059 deaths from consumption and 105,971 from pneumonia. A few other figures were, in round numbers, as follows: Diarrheas, 47,000; typhoid, 35,000; cholera infantum,

26,000; influenza, 17,000; diphtheria, 16,000; whooping cough, 10,000; septicemia, 7000; scarlet fever, 6000; meningitis, 4000.

Pneumonia of late years seems to be on the increase. American statistics give a rise as follows: Proportion of total mortality, in 1870, 7 per cent.; in 1903, 15 per cent.; in 1904, 19.5 per cent. This must have been due in part at least to influenza epidemics.

Osler states that pneumonia is the most fatal of all acute diseases, killing more than diphtheria, and outranking even consumption as the chief cause of death. Pneumonia remains practically the only infectious process, the incidence and mortality of which is still unaffected by the fact that its etiology is well understood, and by that education of the public in health matters which is producing such excellent results in other infections, such as tuberculosis and typhoid fever. It is perhaps a reproach, and certainly a cause of chagrin to our profession that in spite of a hundred years of general progress, and many and varied methods of treatment for it, the mortality of pneumonia stands unabated.

Its incidence among the urban population is distinctly greater than among the rural. Its seasonal variations correspond closely with those of other respiratory diseases, such as bronchitis and influenza, being greatest during the first four months of the year; although pneumonia, especially as a complication of other conditions, such as typhoid fever or septicemia, may occur at any season.

Etiology. The influence of season has already been referred to. While all ages are liable, pneumonia is most prevalent, particularly in the acute lobar form, in early middle life. While not infrequent in little children even before two years of age, the patchy type is common, although the statistics for these early ages are somewhat vitiated by the difficulties of diagnosis, and what begins as a lobular may end anatomically as a lobar type of the disease, by coalescence of areas of inflammation. Cases rapidly become more numerous up to the age of thirty years. It should be borne in mind, too, that specific fevers, such as measles, diphtheria, and typhoid are much commoner up to this age, and more frequently accompanied by what is called pneumonia, while influenza has a vastly greater effect than any other specific fever upon the frequency and fatality of pneumonia.

As regards sex, males suffer in the proportion of about two to one female, the difference being explained partly by the greater exposure of males in their occupations, the influence of which must be admitted as a matter of universal experience. Habits, too, of alcoholism and other forms of excess have their bearing, although good authorities state that alcoholism *per se* has no special effect in determining the attack all admit that it enormously increases the fatality, over 60 per cent. of intemperate persons who are attacked dying.

CONTAGION AND INFECTION. The questions of contagion and infection are not yet settled. So-called epidemics of pneumonia are really only local outbreaks due to the same cause in each case, or determined by the incidence of other diseases, particularly influenza. Still, I fancy, none of us has failed to observe on occasions a series of cases arising in the same house, apparently clearly from the first case. Measures of isolation such as are applied to infective diseases have never been hitherto accepted as necessary. More important is the bearing of this question upon the problem of immunity. It is universally admitted that one attack predisposes to another, so that immunity, if conferred at all, is very short-lived, as in diphtheria, where the average period of immunity is fixed at three weeks, so that all efforts at preventive inoculation have so far proved failures. Every successive attack is more dangerous to the sufferer than those that precede. Three, four, and five recurrences are not very rare. and instances of recurrence, even to the twenty-eighth and thirtieth attack have been recorded. Another line of investigation and treatment is that by serums, as in the case of diphtheria. The antitoxin serum as employed first by Klemperer has been actively followed up, but so far without the success for which one might hope. The sera employed have been those of the sheep, the cow, the ass, and the rabbit, as well as that of persons convalescing from pneumonia. There have also been employed the expressed juices of pneumonic tissues, and the filtered and sterilized products of pneumococcus cultures; but the system has not reached what we may call a commercial basis, as in the case of diphtheria, and one is justified in saying that so far the serum treatment has not realized the promise which it at first held out.

Without unduly anticipating as to treatment, it seems fair to say that the vaccine treatment shows more promise of success, particularly in cases of unresolved pneumonia, and in any of the numerous extrapulmonary local infections, such as pleurisy or empyema, pericarditis, endocarditis, peritonitis, otitis, synovitis, meningitis, etc., or in the rare case of generalized infection, the so-called pneumococcus septicemia, particularly where there is no lung lesion.

Many cases reported by competent observers also seem to justify the statement that, as a rule, only an autogenous vaccine is of service, the strains of the organism being so many and varied that stock vaccines are not likely to be of service. A writer in the *British Medical Journal* of June 26, 1909, concludes as follows: (1) Successful inoculation for pneumonia is possible. (2) It does no harm. (3) The vaccine used should be autogenous if possible, if not, it should be from one or more strains known to be virulent. About six days usually suffices to attenuate a virulent strain of pneumococcus. (4) It should be used early. (5) The estimation of the opsonic index is not necessary.

The usual dose is 10,000,000 to 50,000,000 organisms, repeated every two to even ten days, according to clinical indications. I venture to add that vaccines should not entail the nonuse of other approved lines of treatment.

Morbid Anatomy. It would be a waste of time to go over this familiar ground, but one may note a few outstanding points.

The absence of relation between the size of the lesion and the severity of the constitutional disturbance. One frequently sees a case with a consolidation no larger than a tangerine orange, and very severe fever and other reactions, while another case with double lesions, involving perhaps two-thirds of the lung area, may have much less fever, delirium, and other such disturbances. This may be due to differences in the virulency of the strains concerned, a marked feature of the pneumococcus.

Another very important point in the morbid anatomy is the degree of associated bronchitis. The bearing of this upon such points in treatment as the use of morphine is, as we all know, all-important, and as to prognosis, too much stress cannot be laid upon the fact that bronchitis clearly complicates the outlook, particularly if it has developed in the course of pneumonia. If the pneumonia arises in the course of a preceding bronchitis, or "cold in the chest," the latter is not necessarily so serious a complication. It may be of interest to remind ourselves that the healthy lungs in adults weigh, for the male: right, 24 ounces, left, 21 ounces, total, 45 ounces; for the female: right, 17 ounces, left, 15 ounces, total, 32 ounces. Extraordinary alteration in these weights is observed as a result of pneumonia. Even the sound lung, on account of hyperemia and compensatory change, is heavier than normal, while the consolidated lung has been found, especially the right, to weight over 100 ounces.

With regard to the parts of the lung usually attacked, the order of frequency is (1) the right base; (2) the left base; (3) the right apex; and (4) the left apex, apical pneumonia being relatively much more frequent in children than in adults.

Pathological Results. We may speak of these as death, or resolution more or less complete. In regard to death, it is only comparatively recently that we have realized that death occurs usually not directly because of the condition in the lung, but through the cardiovascular apparatus. Perhaps in some cases the patient may be said to die of exhaustion or asthenia or severe toxemia, the cause of death lying in the higher centres of respiration and circulation; and it may be admitted that other cases die, at least indirectly, because of such complications as tympanites. The latter condition should be looked upon as a complication, in so far as it embarrasses the diaphragm and the heart; but it is even more significant as a symptom of general neuromuscular failure, and is accompanied often by other evidences of the same thing, such as capillary stasis and low muttering delirium.

With regard to the cardiovascular apparatus, the study of the blood pressure has given us valuable information. G. A. Gibson, of Edinburgh, in 1908, in the *Edinburgh Medical Journal*, and G. A. Gordon, in 1910, in the same journal, have shown us that if the blood pressure in millimeters of mercury falls below the pulse rate per minute, recovery is very unlikely. This disturbance of ratio between blood pressure and pulse rate may be looked upon in two ways, and the bearing of the point upon treatment is very important. We have been accustomed, of course, to study this question by watching the first sound of the heart, the second pulmonary and aortic sounds, the pulse, and the degree of dyspnea and cyanosis, and our anxiety rises as these untoward signs advance; but a rapidly rising heart may be due to the attempt to keep full a set of arteries in which blood pressure is rapidly declining. In such cases the use of caffein, digitalis, strophanthus, strychnine, etc., is urgently called for, while the use of a nitrite would only tend to further handicap Nature's efforts. On the other hand, a heart rate rapidly rising without a fall of blood pressure probably means not a vascular but a cardiac defect, the heart muscle being overpowered by high temperature, toxemia, and particularly by previous disease of the heart, muscular or valvular, which always adds greatly to the patient's danger. In such a case nitrites might be of service if the arterial tension is at such a level as to permit of a lowering that would reduce the burden with which the heart is struggling.

Resolution more or less complete, the other pathological result referred to above, fortunately, is complete in the great majority of cases. Incomplete resolution does undoubtedly occur, for reasons hard to explain; but empyema and all other such results are rarely if ever due to a simple pneumococcus infection. Its greater frequency in children is due to their greater tendency to double infection, and a pneumonia of mixed or doubtful origin is more frequently the cause of empyema in the adult. Such a result as chronic interstitial or indurative pneumonia is perhaps never due to simple pneumonia, but in some cases spreads in from the pleura, following its trabeculæ inward, or begins similarly at the hilum of the lung from chronic inflammation of the glands, interstitial connective tissue growth proceeding after the manner with which we are more familiar in the case of cirrhosis of the liver or of the kidney.

In regard to pulmonary abscess and gangrene, while everyone admits that these conditions may follow on pneumonia, they are always due to secondary infection and probably to a preëxisting one, due to chronic bronchitis, sometimes with bronchiectasis, so that the primary causal connection of pneumonia with these conditions is, in many cases at least, distinctly open to question.

One other point in regard to the clinical course, the pulse-

respiration ratio is of great importance. Normally it is about 4 to 1, 72 pulse beats to 18 respirations. At the outset of the disease, one's suspicion of pneumonia is strongly aroused by observing that this is changed, sometimes even to 2 to 1, pulse rate under 90, breath rate 36 to 40. I need not remind you that dyspnea may be accompanied by either slow or rapid breathing. If obstructive, as in croup or asthma, or from a foreign body in the larynx, the rate is apt to be slow; but I am accustomed to teach my classes that wherever vesicular tissue is affected the breath rate will be quickened, hence the great significance of a breath rate persistently slightly raised, say 22 to 24, in early phthisis. The same holds true much more in regard to pneumonia. Only recently a sthenic case in a young Italian in Ward 6, General Hospital, showed a pulse throughout under 90, with a breath rate up to 40; and more recently still in a profoundly toxic case in Ward 35, General Hospital, whose crisis came only on the thirteenth day, the ratio actually stood once at 88 to 64, or nearly 4 to 3; and though the radial pulse remained good throughout, the first sound of the heart was always faint and poor, a discrepancy of signs for which I had in his case no explanation to offer, as he had no pericardial effusion or other discernible cause for the faintness of the heart sounds, and I cannot think that it could have been due to myocardial degeneration, from the general good conduct of the organ throughout the illness.

I need not say that as a point in prognosis this is most important —not that the mere disturbance in ratio is so important—but that so long as the pulse remains below at most 120, it makes little difference how high the breath rate goes.

PROGNOSIS. Habits and previous state of health: In any given case, habits as regards alcoholism and other excesses may perhaps be mentioned as first in importance as affecting the outlook.

Heart: Perhaps second in importance, if not first, is the state of the heart as to previous disease, muscular or valvular. While many such cases do recover, their danger is immensely greater.

Age: The mortality tables show after five years of age a steadily increasing risk as life advances.

It is of the utmost importance to discriminate between a complicating and a primary pneumonia. Inflammation of the lung arising in the course of influenza, typhoid, apoplexy, or any other disease is much more apt to prove fatal than a pneumonia which is itself the primary disease.

The condition of the non-pneumonic parts of the lung: Where congestion, bronchitis, and edema remain absent from the other parts of the lung the original pneumonic area may become very extensive without seriously jeopardizing life; and the converse is true, that a case with even a small pneumonic area may be fatal if accompanied by extensive irritation in the other areas of the lungs.

We have already referred to the prognostic value of the ratio between pulse rate and blood pressure.

The limitations of general private practice do not ordinarily permit of a white cell count, although we have known for years that a marked increase is of good prognostic significance, while cases in which this fails to occur often die. I am accustomed to teach my classes that where the microscope cannot be used, other almost equally certain evidence of good reaction and good prognosis are as follows: (1) Early and well-defined herpes labialis. This occurs in from 13 to 43 per cent. of cases, according to various authors, and is much more frequently seen with pneumonia than with any other febrile disease. It is sometimes delayed to the fifth or even the seventh day, and if very pronounced and pustular, may be an evidence not of good but of lowered resistence. (2) Sharp and early high temperature, well maintained. (3) The early expectoration of sputum, highly fibrinous, viscid, rusty of course, but not too bloody, and particularly not too fluid, which indicates absence of fibrin and poor reaction. Some at least of these three points, I think, will be found usually to coexist with marked leukocytosis.

Marked neuromuscular relaxation and failure of the nervous system to retain control of the unstriped muscle tissues are signs of gravity. They are shown by the onset of tympanites, of hyperresonance, and relaxation of other portions of the lungs, and of bronchitis or edema. Especially if associated with low delirium and dry, brown tongue these changes are of serious import.

One may mention finally, not exactly as a curiosity, the loss of the knee-jerk, first referred to in 1894 by that prince of clinicians, Dr. Hughlings Jackson. This reflex disappears usually on the third or fourth day, returns about the ninth day, slightly increased, and becomes normal about the end of the second week in ordinary cases. It has no relation to the crisis, and its disappearance before the third day means danger; while if it remains normal until the seventh day recovery may be considered practically certain. Even in diagnosis this may serve to distinguish tuberculous and other non-pneumococcal pneumonias, in which it usually persists, or is increased.

TREATMENT. I now come to what is after all the most important part of the whole subject, namely, "treatment." Perhaps no disease has been treated in ways so various by what one might almost call differing schools of treatment. Thus we have: (1) What one might call the expectant school, who prefer to wait, leaving everything to nature until she is evidently in difficulty, when some symptom arises which urgently requires relief. (2) Another group is composed of those who believe that early measures are important and who launch out at the outset with very active treatment, believing perhaps that the disease can be aborted or

the oncoming area of consolidation lessened. These two schools stand at the opposite poles of professional opinion. (3) A third group, and one of whom little but bad can be said, might be called "the specific group," who treat every case on the same plan or with the same remedies, ignoring not only the differences in the personal equation of the patient, but the fact that even as a disease pneumonia is subject to very great variations in virulency and therefore in symptoms. (4) A fourth group might be named "the nihilists," who leave everything to nature, as if they believed that any interference was useless or even harmful.

In regard to these groups, one may say that no intelligent physician would nowadays consent to belong to any one of them. About the last group, one may say that it can be only bad policy to make it a rule to do nothing in every case, while it is quite true that many cases do very well with careful watching and practically no treatment.

In regard to the third group, it is almost as unintelligent to have a single plan or a single drug like digitalis, and yet one occasionally hears papers read and discussed at medical meetings by practitioners of this type, of whom it may almost be said in the words of Holy Scripture, "He openeth his mouth and saith unto everyone that he is a fool."

In connection with the second group, it seems to me that there is more reason in their method, particularly if regard be had to the value of such measures as are meant to reduce the toxemia and fever at the outset, such as free purgation, diaphoresis, and diuresis, assuming, of course, that the case is a sthenic one, and seen early.

The views of the opposing schools, if one may call them so, can never be reconciled by appeals to statistics, as it is impossible to obtain a series which differs only in the line of treatment adopted. They differ also in other respects impossible to estimate, such as age, habits, station in life, previous health, season, place in which they live, severity of infection, individual power of reaction, the particular strain of pneumococcus; and the public must trust to the experience, judgment, and common-sense of the physician whom they select.

Let us therefore see what are the main indications in the disease, as well as in each individual case, and this can best be seen by considering how and why death occurs in fatal cases. In most such cases the causes of death are three, namely, toxemia, cardiac failure, or some complication.

A rational treatment, therefore, should have in view the obviating of these three conditions, to which one may add a fourth, the limiting of the lesion in the lung.

Toxemia. There seems little doubt that the few cases that were benefited by the old heroic treatment of bleeding at the outset may have been so helped by the lessening of toxemia by abstraction

of blood, which may also have tended to limit the pulmonary lesion mainly by making the blood less fibrinous. But more modern methods of treatment seem to relive toxemia by free and early catharsis, for which calomel and salines or compound jalap powder are in my judgment best, along with free diuresis. The latter is best obtained by abundant water and perhaps light salines such as citrates, the calomel, of course, acting in the same way. As to diaphoresis, hydrotherapy in the form of hot packs and mustard foot baths are safe and simple methods of securing free skin action, if it be thought necessary.

Cardiac Failure. This can best be prevented only if we clearly understand what is most likely to cause it. Toxemia and fever are the most important causes. Abdominal distention, as everyone knows, is much to be feared for this reason, and the feeding, therefore, should be carefully considered. It seems to me that an excess of carbohydrates and of milk, especially if the patient is rendered flatulent by the salines above mentioned, should be avoided. Constant pain, too, tends to induce cardiac failure, and one of the worst features in the disease is restlessness and delirium. Simple wakefulness is dangerous, and should be controlled by morphine if necessary, but preferably by paraldehyde, 1 to 2 drams, or ammonium or sodium bromide, 40 to 60 grains, both of them administered preferably per rectum. Strychnine should be given before cardiac failure threatens, because in addition to being a good and prolonged cardiac stimulant it is a general tonic. It keeps the respiratory centre awake, and tends to prevent abdominal distention.

In regard to digitalis, while not ignoring the results of laboratory research, rather being grateful for our added knowledge of its pharmacology, this is a case in which one may depend only on clinical evidence as to its value, and it was found to be useful in this disease before its dangers in other acute infections were recognized.

I am accustomed to teach my classes that the use of digitalis presupposes a heart muscle which is histologically sound. Its use is therefore dangerous in such diseases as erysipelas, scarlet fever, septicemia, typhoid, in which usually the patient has been a long time ill before the heart gets into serious difficulty, when cloudy swelling or other degeneration has been set up, which makes the drug dangerous. Cardiac failure in pneumonia, however, if it occur at all, shows itself within eight or ten days, within which time serious muscle change can hardly have occurred; hence, I believe, its great usefulness when needed in pneumonia. If the stomach is acting badly, or the tongue dry and brown, it had better be given hypodermically.

Oxygen, while probably more useful in bronchopneumonia or capillary bronchitis than in true pneumonia, has its place as a cardiac stimulant, and probably assists in reducing toxemia.

The indications for alcohol in this as in any other acute infection are, I think, the presence of two or more of the following conditions: (1) Dry brown tongue; (2) marked delirium, especially of the low muttering type; (3) a rapid and rising heart rate; (4) high temperature.

I would oppose its routine use, but where indicated as above it should be used, and if used at all, pushed boldly for twenty-four hours, after which if no improvement be noted, it had better be gradually withdrawn and caffein or perhaps camphor dissolved in oil substituted.

OPIATES. The use of opium in any form constitutes one of the most important questions in the management of this disease. My own rules about it are as follows: (1) Even minute doses are dangerous at either extreme of life. (2) Use it in no case in which there is much bronchitis, edema, or other abnormal condition of the non-consolidated portions of the lung. (3) Avoid it when marked cyanosis exists, especially with pallor and sweating, and tympanites.

The main conditions calling for an opiate are severe pain, marked sleeplessness and restlessness, and threatening cardiac failure. In the latter case, very small doses are best, say gr. $\frac{1}{32}$ repeated.

Pain, for instance, can usually be controlled by the poultice or ice bag, preferably the latter in the adult, or by leeches, and it at any rate usually occurs early if at all when one or two doses of morphine are less likely to be harmful. The drug tends to favor toxemia by locking up the bowels, which must be guarded against, as already indicated, by purgatives.

COMPLICATIONS. In regard to complications, one can best foresee and guard against these by having a clear conception, to be obtained only by experience, of the average standard case of pneumonia. The composite photograph carried in one's mind, the result of careful study of perhaps 100 cases, would lead one to expect a temperature of 103° F., a pulse less than 120, and a breath rate less than 40. Any departure from this ratio should be carefully watched.

For the control of fever, cool air, light clothing, the use of the ice bag or cold sponge in adults, the hot pack in children, should suffice. Antipyretics should rarely if ever be used, as transient in their effects on the fever and depressing to the heart; and of course nothing in the treatment should be permitted which tends even remotely to induce cardiac failure. I cannot, however, refrain from mentioning a series of cases of acute pneumonia which I once saw in camp with the 5th Battalion, the Royal Warwickshire Regiment, in England. They were all strong young sthenic cases and they lay on stretchers in an open marquee in hot weather in August, with nothing but milk and water to drink, and one might almost say phenacetin to eat. They all made excellent recoveries. Under

ordinary conditions such treatment would be impossible. It must be remembered, also, that moderate fever is a reparative process meant to stimulate leukocytosis and phagocytosis, and constituting a most important phase of the reaction to the infection. Quinine in single doses, 20 to 30 grains, is not depressing, although sometimes nauseating; and alcohol, as above mentioned, serves to spare tissue waste when fever is high. Other complications, such as delirium, abdominal distention, and declining blood pressure, have already been noted, and in this paper cannot be referred to at greater length. Neither is it within the scope of this article to discuss such complications as pericarditis, empyema, or other pleural effusions, lung disease or gangrene, meningitis, neuritis, arthritis, or phlebitis; each calls for its own special treatment, and while bearing in mind that the case is still one of pneumonia, one should neither lose hope nor abandon treatment because of the appearance of any of them.

It is superfluous to emphasize our modern views as to the necessity of fresh air, proper restriction of dietary, and the non-necessity of the poultice except for pain. Popular prejudice, of course, especially in private as against hospital practice, often denies us a free hand in the nursing details, as we all know that on many occasions when the patient dies the open window and the ice-bag, or underfeeding, or overuse of alcohol, will all be blamed by the friends as the cause of death.

Finally, with regard to convalescence: let me remind you of the dangers that attend the crisis, particularly in the aged. How often has one been grievously disappointed, especially in the case of the old, by death from cardiac failure just after the storm seems to have been weathered; and even in a sthenic case, where a critical fall of temperature is not accompanied by much danger to the heart, and the marked return of the sense of *bien-être* has made the patient anxious to be early out of bed, it is well to insist upon at least ten days' convalescence in bed: (1) To allow the heart to recover its proper tone; (2) to avoid risk of empyema, especially if there has been extensive pleurisy, and to allow complete resolution, for the lung tissue remains tender for at least that length of time after the patient's strength has begun to return.

In conclusion, it will be seen that although at too great length, I have not attempted to treat the subject exhaustively.

THE ANATOMICAL EXPLANATION OF THE RELATIVELY LESS RESONANT, HIGHER PITCHED, VESICULOTYMPANITIC PERCUSSION NOTE NORMALLY FOUND AT THE RIGHT PULMONARY APEX.[1]

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Most clinicians, especially those who have devoted particular attention to diseases of the lungs, are agreed that in percussing the upper parts of the chest a slight difference in sound is normally heard when the two sides are compared. This difference consists in the fact that on the right side, from the apex down to about the level of the second interspace or the third rib, the note elicited is slightly higher in pitch, shorter in duration, and less resonant. Occasionally, also, the note is somewhat more tympanitic in quality.

The actual amount of difference in the afore-mentioned sound attributes, while never great in degree, is of extreme clinical importance on account of the frequency with which incipient tuberculosis manifests itself by physical signs at the right apex. If no allowance be made for a normal variation in the percussion sounds of the two sides of the chest, errors in diagnosis easily may occur.

On consulting various text-books on the subject one finds that in reference to the question under considertaion they may be divided into four classes: (1) Those in which allusion is made to the difference, but no explanation is offered. (2) Those in which no mention of any difference is made. (3) Those in which it is denied that any difference exists. (4) Those in which more or less explanation of the phenomena is attempted.

The following statements are taken from works on physical diagnosis which comprise the first class:

"On the right side the sound is somewhat less clear, shorter, and of a higher pitch than on the left." (J. M. DaCosta.[2])

"The pulmonary quality is slightly less marked on the right

[1] Read before the College of Physicians of Philadelphia, January 3, 1912.

[2] Medical Diagnosis, 1900, p. 270.

than on the left in supraclavicular and infraclavicular areas." (G. R. Butler.[3])

"It [the resonance] is slightly higher in pitch at the right than at the left apex." (J. H. Musser.[4])

Similar statements are made by J. C. Wilson[5] and R. C. Cabot.[6]

Among the authors who make no mention of the normal variation in the percussion note of the two sides are: Loomis,[7] Gibson and Russell,[8] Vierordt,[9] J. H. Bass,[10] P. Simon,[11] S. Gee,[12] A. Weil,[13] P. Niemeyer,[14] Ebstein,[15] Stokes,[16] Sahli,[17] Hare,[18] Fowler and Godlee,[19] and Fuller.[20]

In the third class we found only one book, that by S. G. Bonney,[21] who says, "The characteristics of normal resonance are identical upon the two sides of the chest in this (infraclavicular) region, as contrasted with the striking differences displayed below the third ribs."

The text-books of the fourth group offer numerous explanations of the phenomena.

The explanation is based on the thickness of the pectoral muscles by the following writers: A. Flint,[22] J. G. Brown,[23] Edlefsen,[24] Beevor,[25] Guttman,[26] E. Cassaët.[27]

Eichhorst[28] says that the difference is most marked in men of laborious occupations. He goes on to say, however: "Bei Linkshaendingen habe ich den Perkussionsschall beiderseits laut gefunden."

Brugsch and Schittenhelm[29] and C. L. Greene,[30] regard the difference as due to the nearness of the large bronchus.

Kidd[31] attributes the variation in part to the difference in the

[3] The Diagnostics of Internal Medicine, p. 426.
[4] A Practical Treatise on Medical Diagnosis, 1899, p. 495.
[5] Medical Diagnosis, 1909, p. 131.
[6] Physical Diagnosis, 1909, p. 130.
[7] Lessons in Physical Diagnosis, 1887, p. 27.
[8] Physical Diagnosis, 1902, p. 142.
[9] Lehrbuch d. klin. Untersuchungsmethoden, 1904, i, 112 and 634.
[10] Medizinische Diagnostik, 1883, p. 112.
[11] Man. de Percussion et d'Auscultation, 1895, p. 35.
[12] Auscultation and Percussion, 1907.
[13] Handbuch u. Atlas der Topograph, Perkussion, 1877, p. 42.
[14] Handbuch d. theor. u. klin. Perkussion u. Auskultation, 1870, p. 130.
[15] Leitfäden d. Aerstlichen Untersuchung, etc., 1907, pp. 151 to 158.
[16] Diagnosis and treatment of the Diseases of the Chest, 1837, Part 1.
[17] A Treatise on Diagnostic Methods of Examination, 1905.
[18] Practical Diagnosis, 1906.
[19] Diseases of the Lungs, 1898.
[20] Diseases of the Lungs and Air Passages, 1867, p. 51.
[21] Pulmonary Tuberculosis, Philadelphia, 1908, p. 183.
[22] Variations of Pitch in Percussion and Respiratory Sounds, 1852, p. 9.
[23] Medical Diagnosis, Edinburgh, 1887.
[24] Lehrbuch d. Diag. d. inn. Krankheiten, 1889, p. 449.
[25] Lancet, May 4, 1901, p. 1275.
[26] Lehrbuch der Klin. Untersuchungsmethoden (Klemperer), 1904, p. 145.
[27] Precis d'auscultation et de Percussion, 1906, p. 563.
[28] Lehrbuch d. Physikal. Untersuchungsmethoden, Berlin, 1889, i, 269.
[29] Lehrbuch Klinischer Untersuchungsmethoden, 1908, p. 80.
[30] Medical Diagnosis, 1910, p. 101.
[31] Allbutt's System of Medicine, 1909, v, 346.

bronchi, stating that the right main bronchus is slightly wider and more vertical, and that the bronchus to the upper lobe is given off higher up, that is, near to the trachea.

In answer to this it may be stated that the point at which the upper lobe bronchus of the right side is given off is opposite the upper border of the third costal cartilage. Its effect on resonance at the apex must be very slight.

Walshe[32] states that "In a certain number of persons whose lungs are perfectly sound the right infraclavicular region gives a more wooden note under percussion, and is at the same time less resonant than the left. This peculiarity is perfectly unconnected with excess of muscular substance on the right side, and depends on some hitherto unascertained condition."

In another place[33] the same author writes that: "Numerous differences in the results of percussion of corresponding points of the two sides of the chest have been noticed, and traced to an obvious cause the presence of texture and organs of different densities in those spots. Variable thickness of the external soft parts will have a similar effect; thus the right infraclavicular region is less resonant than the left in robust persons whose employment requires much use of the right arm; the pectoralis muscle enlarges from use. Here the explanation is obvious, but the explanation is not obvious when the right infraclavicular region emits less tone than its fellow, in persons presenting no muscular thickening of this kind. Such inferiority of resonance (sometimes attended, too, with slight elevation of pitch) is never great provided the lungs are perfectly healthy; it holds good whatever be the direction given to the fingers, and whether they fall outward toward the humerus or inward toward the sternum."

H. S. Anders[34] attempts an explanation by stating that in addition to greater thickness of the pectoral muscle and the presence of the liver on the right side, "an explicit reason is found in the fact that there is a different anatomical arrangement of the bronchial tubes, those on the right side being larger and situated more superficially and higher up; this, with a bunch of medium and small branches occupying space, that on the left side is filled with air vesicles, gives an amount of bronchovesicular tissue sufficient to elevate the pitch slightly but perceptibly, the tubes adding an element of tympany, the extra muscular and connective tissue of their firm walls an element of dulness."

Anders and Boston[35] state: "The higher pitched percussion note over the right than over the left apex is probably due to larger diameter and higher position of the right bronchus. The note is again modified by the thickness and tension of the chest wall as the result of muscular contraction, etc."

[32] Practical Treatise on the Diseases of the Lungs, 1871, p. 64.
[33] Lancet, 1849, i, 196.
[34] Physical Diagnosis, 1907, p. 99.
[35] Text-book of Medical Diagnosis, 1911, p. 55.

Gerhardt[36] offers the following reasons: (1) Increased size of the muscles on the right; (2) slight scoliosis often present; (3) difference in anatomical structure: (*a*) bronchi larger, (*b*) pulmonary apex smaller and lower, (*c*) bronchi retain large caliber near to the surface.

TABLE OF RESULTS OBTAINED BY EXAMINING THE CHESTS OF THIRTY-TWO LEFT-HANDED INDIVIDUALS.

No.	Circumference of chest (inches)		Vocal fremitus		Percussion resonance		Pitch (higher)		Breath sounds (harsher)		Remarks
	R	L	R	L	R	L	R	L	R	L	
1	17¼	17	+	..	−	..	+	..	Equal		
2	14½	14	+	..	−	..	+	..	Equal		Poorly developed; 18 yrs. old; looks to be 14.
3	16¼	16	+	..	−	..	+	..	+	..	
4	18¾	18	+	..	−	..	+	..	+	..	Large deep pectoral muscles; difference only on deep percussion.
5	16	15¼	+	..	−	..	+	..	+	..	Large left pectoral muscles; difference slight.
6	16½	16½	+	..	−	..	+	..	+	..	Well marked difference.
7	17½	17¾	+	..	−	..	+	..	+	..	
8	16½	16¾	+	..	−	..	+	..	+	..	
9	15¾	16¼	+	..	−	..	+	..	+	..	
10	16	16	+	..	−	..	+	..	+	..	Bilateral subclavian murmur.
11	15½	15¾	+	..	−	..	+	..	+	..	Difference slight. Funnel breasted.
12	17¾	17½	+	..	−	..	+	..	+	..	
13	15½	16	+	..	−	..	+	..	+	..	Marked retraction of whole right side.
14	16½	16½	+	..	−	..	+	..	+	..	
15	17¾	17½	+	..	−	..	+	..	+	..	
16	16	16	+	..	−	..	+	..	+	..	
17	18	17	+	..	−	..	+	..	+	..	
18	17	16½	+	..	−	..	+	..	+	..	
19	16½	17½	+	..	−	..	+	..	+	..	
20	17¾	18¼	+	..	−	..	Equal		+	..	
21	16½	18	+	..	+	..	+	..	−	+	Flat left chest; looks pathological.
22	18¼	18½	+	..	−	..	+	..	+	..	
23	15½	15¼	+	..	..	+	..	+	Equal		Very poor expansion; feebie breath sounds.
24	18½	18½	+	..	−	..	+	..	Equal		
25	18¾	18	+	..	−	..	+	..	+	..	
26	17	17	+	..	−	..	+	..	+	..	
27	16½	17	+	..	−	..	+	..	+	..	
28	15¾	15¾	+	..	−	..	+	..	+	..	
29	16	15¾	+	..	−	..	+	..	+	..	Difference reversed on light percussion.
30	18½	18	+	..	−	..	+	..	+	..	
31	18	17½	+	..	−	..	+	..	+	..	
32	16¾	16¾	+	..	−	..	+	..	+	..	

[36] Krause's Lehrbuch d. klin. Diagnostik innerer Krankheiten, 1909, p. 122.

Inasmuch as none of these explanations are adequate to account for all cases, we have endeavored to solve the problem by the careful anatomical study of frozen sections of the cadaver, previously hardened in formalin, and by a critical study of the chests of 32 left-handed students. The examinations of the left-handed individuals were made with especial reference to the following points: (1) Circumference of the chest, right and left. A tape-measure was applied to the side of the chest, extending from the middle of the spine to the mid-sternum at the level of the nipple. For obvious reasons errors in such measurements may easily be made, and we, therefore, lay no great stress on the importance of the data thus obtained. The results show, however, that the *right* chest is not infrequently larger even in left handed-persons. (2) Vocal fremitus, elicited below the clavicle. (3) Percussion resonance, from the clavicle to the upper border of the third rib. (4) Percussion pitch, elicited over the same area. (5) Character of the breath sounds in the same region. Corresponding points on the two sides of the chest were of course compared, and the results immediately recorded.

The foregoing table represents in charted form the results of our examinations, and the data obtained were so uniform that it seemed needless to examine a larger number of men. From these studies the following conclusions may be drawn:

Vocal fremitus is normally more intense on the right side, even in left-handed individuals, and probably is due, as was pointed out by one of us (Fetterolf[37]), to the immediate contact of the trachea to the right pulmonary apex. On the left side the aorta, the common carotid and subclavian arteries, and the esophagus, together with lymphatic and areolar tissue, a layer some 2 to 3 cm. in thickness, are interposed between the lung and the trachea.

While studying the percussion sounds at the two apices it was occasionally noted that after prolonged percussion differences quite noticeable at first tended to become less marked. This is by no means an original observation, and may be explained as due to one or all of the following factors: (1) Aural fatigue on the part of the examiner. (2) A pulmonary reflex, as described by Abrahms, by virtue of which percussion over a certain area of lung tissue produces a local reflex dilatation of the air vesicles, thus obscuring a slight degree of dulness. (3) Spasm of the deeper fibers of the pectoral muscle, analogous to the increased myotatic irritability of myoedema seen in low nerve tone,[38] which by increased muscular density tends to limit the penetration of the percussion vibrations.

In persons of poor muscular development, in case of the aged, the wasted, and the muscularly inactive, the difference in the

[37] Arch. Int. Med., February, 1909.

[38] This explanation, used in a somewhat similar connection, has been suggested by M. E. Rist, Percussion Thoraccique, La Presse Medicale, December 16, 1911.

pulmonary resonance is present, while a corresponding unevenness in the thickness of the pectoral muscles may be either inconsequential or entirely lacking. The main cause, then, must lie deeper than in the muscles of the chest.

Let us consider for a moment what physical conditions underlie the changes in the percussion note which we are endeavoring to explain, namely, (1) diminished resonance; (2) elevated pitch; (3) a tympanitic element.

1. *Diminished resonance* over normal lung implies a lessened quantity of air and elasticity or the presence of overlying tissue, which by reason of its density vibrates less complexly, and transmits vibrations less well. The result, acoustically speaking, is a condition in which the fundamental note preponderates and fewer overtones are heard. The sound is more muffled and is shorter and weaker, tone enforcement being diminished. With the appearance of dulness there is always an increase in the elevation of pitch.

2. *Elevation in pitch* implies an increased vibratory rate, and this in normal pulmonary tissue again depends largely, other things being equal, on the amount of air, the size of the air columns.

3. *A tympanitic quality* is heard over pulmonary tissue when: (*a*) Air columns vibrate in smooth walled chambers which communicate with the atmosphere; (*b*) when air in closed cavities vibrates in unison with elastic walls; (*c*) when the normal pulmonary tension is relaxed, so that the lung vibrates as a whole (for example, in pleural effusion). Acoustically the underlying characteristic of tympany is preponderance of overtones. For the present purposes only the first proposition (*a*) need be considered.

It may be stated as a general principle that a less resonant note is produced by an increase in the amount of airless tissue. Writers who have hitherto considered this to be a factor, have confined their explanation to conditions of the chest wall, assuming that right-handed people have so much more muscular tissue over the pulmonary apex on that side, that the percussion note becomes dulled in quality. Thus two left-handed individuals were exhibited before the Clinical Society of London by Sir Hugh Beevor to prove this very point, that is, that the left side was less resonant.[39] Similar conclusions were reached by Brazil,[40] who, having studied a number of cases, states that the higher pitched note on the left, in left-handed people affects the entire chest, and is not limited to the apices. How this can be it is difficult to understand, because in the axillary region, for example, muscular development plays practically no role.

Similar views are held by E. Lloyd Jones,[41] who in an interesting article quotes as corroborative evidence the fact that in a right-handed patient of his the left side was less resonant than the

[39] Lancet, May 4, 1901, p. 1275.
[40] Brit. Med. Jour., October 26, 1899, p. 920.
[41] Brit. Med. Jour., October 24, 1903.

right on account of a "former venous thrombosis in the left arm," the veins in the upper part of the left chest being much enlarged. It seems to us that Jones' explanation, which links a diminished resonance with the presence of thrombotic vascular changes on the left side, is in entire consonance with the explanation proposed in this communication. We believe, however, that the percussion changes in such a case as he reports are due more to a distention of the large vessels crossing the apex than to a thickening of the superficial tissues.

AUTHORS' EXPLANATION.

As the result of our studies we believe that the anatomical basis for the conditions under discussion consists of three factors, namely, the anterior position of the vessels in relation to the right apex as compared with the left, the consequent encroachment upon and reduction in size of the right apex, and the apposition of the trachea to the inner surface of the right apex, while the same aspect of the left is in contact with a thick layer of solid tissue.

FIG. 1.—Mediastinal surface of lungs hardened before removal. This photograph shows (*a*) the groove produced in the right apex by the trachea; (*b*) the relatively anterior position of the vessels on the right side; and (*c*) the smaller size of the right apex. *AG*, azygos groove, *VG*, grooves for superior vena cava, innominate vein, and subclavian vessels; *TG*, tracheal groove; *SG*, subclavian groove; *AAG*, aortic groove; *CI*, cardiac impression.

Reduced to their last analysis the anatomical differences between the two sides in the upper thorax will be found to be due to the fact that man is a sinistro-aortic individual. In some of the lower vertebrates, for example, the birds, the aorta is on the right side, and in others, as some of the lizards, there is an aorta on both sides.

In the early human embryo, when the heart is situated far forward in the pharyngeal region, the arrangement of the large arteries is

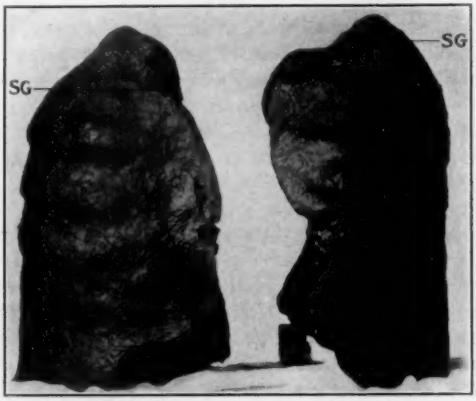

FIG. 2.—Anterior view of lungs hardened before removal. This photograph shows the anterior position of the groove for the subclavian vessels on the right side, compared with the more superior position on the left. *SG*, subclavian groove.

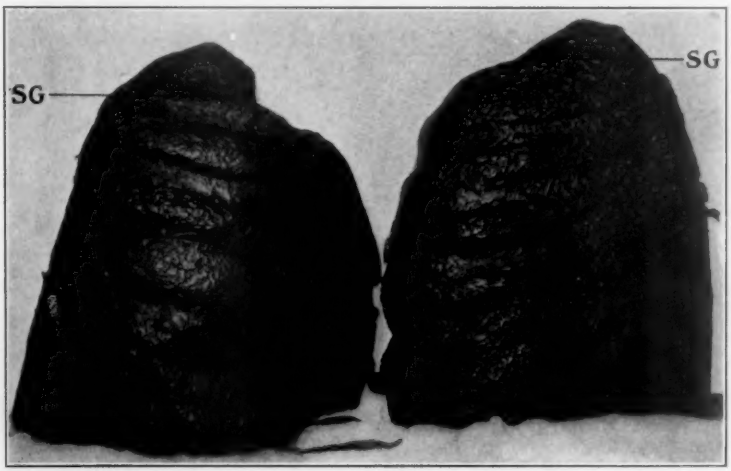

FIG. 3.—Lateral view of lungs hardened before removal. This photograph shows the deeper vascular groove and the smaller size of the right apex as compared with the left.

symmetrical. As development proceeds the main trunk, the aorta, is established on the left side.

In its course from the heart to the vertebral column the systemic aorta passes first slightly to the right, then slightly to the left, and finally almost directly backward between the left side of the trachea and the mediastinal aspect of the left lung. Thus situated it affects both the trachea and the lung, throwing the lower end of the former over toward the right, thereby producing the now well recognized dextro-inclination of the trachea, and grooving deeply, in an arciform manner, the inner aspect of the left lung, in front, above, and behind the root of the latter.

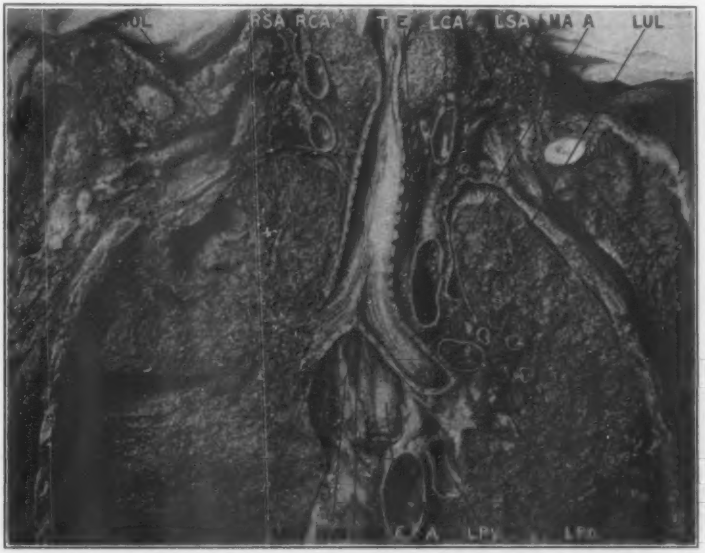

FIG. 4.—Frontal section of the thorax in the axis of the trachea. This photograph shows (*a*) the contact of the right upper lobe with the trachea, (*b*) the mesial position of the left subclavian artery in relation to the left upper lobe, and (*c*) the essentially anterior position of the right subclavian in relation to the right upper lobe. *RUL*, right upper lobe; *RSA*, right subclavian artery; *RCA*, right common carotid artery; *T*, trachea; *E*, esophagus; *LCA*, left common carotid artery; *LSA*, left subclavian artery; *LMA*, left internal mammary artery; *A*, aorta; *LUL*, left upper lobe; *LPV*, left inferior pulmonary vein; *CT*, carina tracheæ; *RVN*, right vagus nerve. (Reproduced from the Archives of Internal Medicine, February, 1909.)

The aortic arch is usually divided by writers into three portions, an ascending, a transverse, and a descending. The first and third of these parts are properly named, but "transverse" is incorrectly applied to the second portion. As regards the aorta, it should be abolished from anatomical terminology and some such term as "horizontal" substituted for it. Its use in this connection probably originated from the fact that when the chest is opened under ordinary condition, the collapsed lungs and the heart fall backward,

and the second portion of the arch becomes approximately transverse. But when the thoracic contents are hardened before the chest is opened it is seen clearly that there is no justification for applying to the second part the term "transverse." When examined under such conditions the general course of the aortic arch is seen distinctly to be almost anteroposterior, there being, of course, a slight tendency to run toward the left. This is demonstrated most clearly in all of the sections that we have prepared; and in nearly all of those cut in the sagittal plane the entire aortic arch from beginning to end is included in the section.

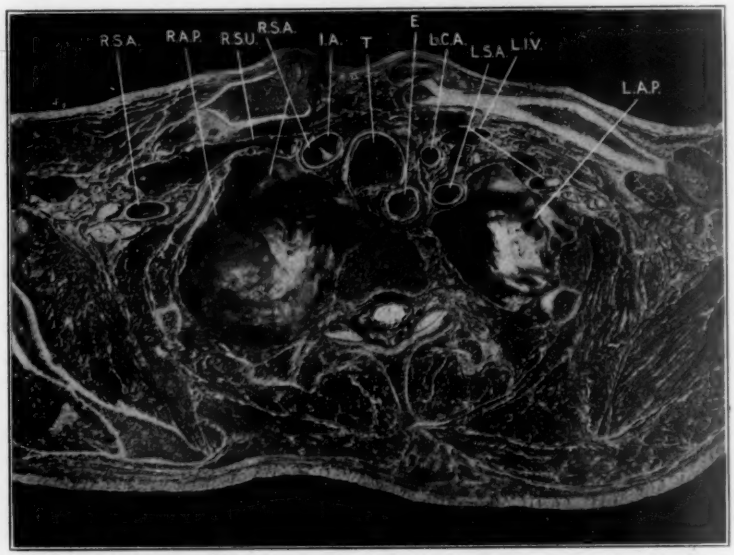

FIG. 5—Section through the upper part of the thorax, viewed from below. The line of section is not exactly horizontal, a slightly lower plane being reached on the right side than on the left. In order to show the apical parietal pleura, the pulmonary apices have been removed. There can be noted in this specimen the beginning contact of the right pleura with the trachea, and the anterior position of the innominate artery, whose bifurcation is well shown. On the left side, the wide separation of the pleura from the trachea by means of the large arteries, esophagus, and areolar tissue can readily be seen. There can be seen well the deep position of origin and the obliquely anterior course of the left subclavian artery. *T*, trachea; *E*, esophagus; *RAP*, right apical pleura; *LAP*, left apical pleura; *IA*, innominate artery, dividing into *RSA*, right subclavian artery and *RCA*, right common carotid artery; *LSA*, left subclavian artery; *LCA*, left common carotid artery; *RSV*, right subclavian vein; *LIV*, left innominate vein.

As stated above, we believe that there are three ultimate factors at work to produce the normal discrepancies at the apices.

First. As regards the anterior position of the vessels of the right side. The first branch, better called the anterior branch, of the horizontal part of the aortic arch is the innominate artery, destined for the supply of the right head and the right upper extremity. Arising, as it does, to the left of the median line, it is compelled to

pass anterior to the trachea in order to reach its destination. This position of its origin is responsible for the anterior situation of the vessels in relation to the right apex. Having crossed the anterior aspect of the trachea and turned somewhat posteriorly, it divides into its terminal branches, the right common carotid and the right subclavian, the former of which does not concern us. The latter passes obliquely upward and *backward* to reach its groove on the upper surface of the first rib. In its course it produces a deep sulcus in the anterior aspect of the pulmonary apex.

The second brach of the horizontal part of the arch is the left common carotid. It passes upward at the side of the trachea, between the trachea and the left apex, and enters the neck.

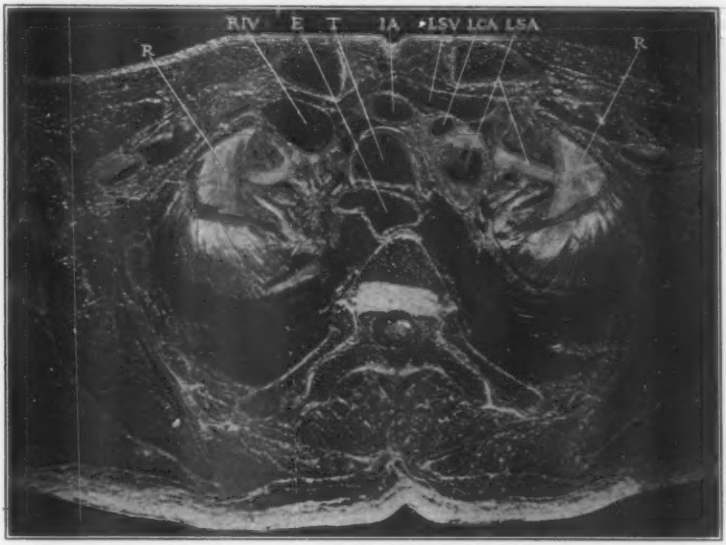

FIG. 6.—Horizontal section of the thorax just above the sternoclavicular articulation. The pleura has been dissected away. This photograph shows (*a*) the anterior position of the innominate artery, (*b*) the posterior position of the left subclavian artery, and (*c*) the anterior position of the right innominate vein. *R*, first rib; *RIV*, right innominate vein; *E*, esophagus; *T*, trachea; *IA*, innominate artery; *LSV*, left subclavian vein; *LCA*, left common carotid artery; *LSA*, left subclavian artery.

The third or posterior branch of the horizontal part of the arch is the left subclavian. This vessel is given off well back in the mediastinum, opposite the posterior surface of the trachea, or even at the left side of the esophagus. It arises so deeply that it is compelled to pass slightly *forward* as well as laterally to reach its groove on the first rib. In the greater part of its thoracic course it occupies the layer of tissue between the left upper lobe and the trachea. Nearing the first rib it produces in the left apex a groove which is shallower and nearer the summit than on the right side.

The same principle applies to the veins. The right apex is encroached upon and markedly grooved by the subclavian and innominate veins, these grooves being continuous with one another, and situated in front of the groove for the subclavian artery. On the left side the subclavian vein produces a sulcus in front of that of the artery, but being more mesial it does not encroach to so great an extent on the apex.

Second. The smaller size of the right apex as compared to the left. This is dependent on the facts enumerated in the previous section. On the right side the vessels pass in front and occupy space which on the left side is occupied by lung tissue.

The shape and size of the pulmonary apices are modified by the vessels in the same way as would be two small hills over which roads were cut. In one case, as on the right side, the road is cut over the side of the hill and requires excavation. On the other side, corresponding to the left apex, the road is carried more nearly over the summit and comparatively no excavation is needed. The result, as far as the apices are concerned, is to cause the former to be smaller than the latter and to give the right apex a more conical, and the left a more dome-like shape.

Third. The apposition to the inner surface of the right apex of the trachea and to the left of solid tissue. This has been emphasized by one of us in a previous paper,[42] and is demonstrated clearly in Figs. 4 and 5.

CONCLUSIONS.

1. The dexterity of the individual has no effect on either the vocal resonance and fremitus or on the percussion pitch and resonance at the apices.

2. The percussion note in the healthy individual is practically always less resonant and higher pitched at the right apex, except in the presence of unusually well-developed left pectoral muscles, and then only if very light percussion is employed.

3. The normal differences are due to the anterior position of the large vessels in relation to the right apex, as compared with the left, to the consequent encroachment upon and reduction in size of the right apex, and to the contact of the inner surface of the right apex with the resonating trachea, while the left is in contact with non-resonating solid tissue.

4. The differences are most marked anteriorly and mesially, because in this situation both trachea and vessels would exert their greatest influence, less marked posteriorly and mesially where the influence of only the trachea would be present, and least marked laterally, since the outer portion of the apex is farthest removed from the trachea, and the vessels at this point are practically similar in their relations on the two sides.

[42] Loc cit.

We wish to express our indebtedness to Dr. George A. Piersol for anatomical material placed at our disposal, to Dr. R. Tait McKenzie for the privilege of examining a large number of left-handed students, and to Dr. P. G. Skillern, Jr., for assistance in preparing our anatomical specimens.

RUPTURE OF THE KIDNEY IN CHILDREN.[1]

By C. L. Gibson, M.D.,

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Ruptures or other subcutaneous injuries are very uncommon in children, only 22 cases being reported in tabulated statistics.[2] My experience comprises 4 cases of complete rupture of the kidney in children, aged from eight to twelve years, and a consideration of the conditions found furnishes some interesting features.

Case I.—Barbara S., aged ten years. Admitted to St. Luke's Hospital August 25, 1902. Two weeks ago was kicked on the right side of the body by a horse; unconscious for a while. Next morning urine contained some blood, none seen since. Some swelling of the right side developed with a considerable amount of pain. Has had no chills, but there have been fever and sweating.

Physical examination showed a bright, healthy child, with a visible swelling of the right lumbar region. No superficial discoloration. The swelling was elastic, insensitive to pressure, flat on percussion. Urine: acid; 1018; no albumin.

Operation: Right lumbar incision, showed the swelling to be a large retroperitoneal accumulation of normal appearing urine. The kidney was ruptured in two, the lower pole entirely separated from the upper three-fourths of the viscus. Nephrectomy; good recovery. Discharged October 3, 1902.

Case II.—These details are as exact as I can furnish them from memory, the record being lost. Boy, aged about ten years. Admitted to the Hudson Street Hospital probably in the summer of 1907. Runover injury; abdominal symptoms; median laparotomy by a colleague; negative findings. Seen by me several days later; diagnosis of rupture of left kidney. Lumbar incision revealed complete tear of left kidney. Nephrectomy; good recovery.

Case III.—James L., aged twelve years. Admitted to the Hudson Street Hospital November, 1909. While running across the street an automobile struck him in the left side, knocking him down. Scalp wound, requiring two stitches. Brought to the hospital in the guilty automobile. Soon began to complain of great pain and tenderness over the left kidney region.

[1] Read before the Section of Surgery of the New York State Medical Society, April 17, 1912.

[2] Watson and Cunningham. Genito-urinary Diseases, vol. ii.

Physical Examination: Tenderness and rigidity in left hypochondrium, also some slight discoloration. Skin and mucous membranes of good color. Shortly after admission passed blood-tinged urine. Hemoglobin, 70 per cent. Operation about eight hours after injury; left lumbar incision; complete rupture of kidney into two pieces. Nephrectomy; drained, good recovery; highest temperature, 100.5° F. Discharged in three weeks.

CASE IV.—M. S., girl, aged eight years. Admitted to St. Luke's Hospital, July 16, 1910. Complained of pain in the "stomach." Two days before she had fallen a distance of four and a half feet, landing on the ground on the right side. Went home; complained of pain in her stomach, which has continued ever since. Bowels regular. No trouble with urination; no blood in the urine; has vomited twice.

Physical Examination: Negative except for the abdomen, which shows general rigidity with tenderness on the lower right side. Temperature, 102° F. Blood count: Leukocytosis, 25,000; polynuclears, 88 per cent. No urine record. Probable diagnosis, appendicitis. Immediate operation. Intermuscular incision; on separating the muscles a considerable amount of fluid was evacuated. On opening the peritoneum a similar fluid escaped from the pelvis. The cecal wall was the site of a considerable ecchymosis. Appendix normal (removed). The wound was dilated retroperitoneally, to allow of a sponge being pushed up into the lumbar region; it returned bloody, but without evacuating any fluid. Injury to the kidney seemed probable; it could be palpated quite readily, but no obvious abnormality being detected (intracapsular rupture) it was decided to await further developments.

The child recovered well and seemed relieved. The urine the next day (July 17): Neutral; 1034; a very faint trace of albumin; a few hyaline casts were present.

July 20. Urine: Acid; 1014; very faint trace of albumin; a few leukocytes.

July 21: Urine: acid; 1020; albumin, 10 per cent.; many red blood cells. In view of this last urine report, exploration was undertaken. Right lumbar incision; the true capsule was found intact, but distended with blood, and raised from the kidney; on opening it, the kidney was found broken completely in two; lower smaller fragment showed beginning necrosis. Nephrectomy; drainage; perfect recovery. Discharged August 9.

This case is interesting, showing a complete rupture resulting from a relatively slight trauma, leaving no mark on the body and producing absolutely no shock, the masking of kidney symptoms by the bruising of the lower abdominal muscles and the colon, the absence of any urinary symptoms until five days after the injury, and also that the kidney may be divided completely in two without appreciable solution of continuity of its capsule.

Four complete ruptures of the kidney in children, aged under twelve years, occurring in the practice of one surgeon seems unusual in view of the small number of such cases on record. It is possible that these cases are really not so rare, and may be overlooked with disastrous results by those who hesitate to interfere in dubious cases. The similarity of the lesions is interesting, being exactly alike in all 4 cases; complete division of the viscus in two parts, the lower one being the lesser. In one instance the capsule remained untorn.

The fact that the kidney lesions were the same with the different kinds of violence seems to confirm the theory of "bursting" by hydraulic pressure. Also the line of rupture; vertical to the long axis at about the junction of the two lower thirds would seem to indicate that we had here an instance of a definite line of least resistance such as I have not seen indicated in any treatise on the subject.

Ruptured kidney. Case IV.

Although the lesion in all these cases was severe, the symptoms, on the whole, were mild, and in several ways deficient. Nephrectomy was necessary in every instance, and successful; no other operation would have been permissible. Three of the children have been under observation and have remained well.

As regards the etiology of such severe injuries, it is obvious that children are relatively little exposed to the various forms of trauma commonly encountered by active men (96 per cent. of all cases). Most modern observations seem to corroborate Küttner's view that the kidney being a semifluid body bursts along the line of least resistance according to the law of hydraulics. Direct pressure from the lower ribs can also explain it. It is less easy, however, to understand the effects of indirect violence, as from a fall on the feet. A point, however, to be borne in mind, illustrated in 2 of my cases, is that the severest form of damage may result from an injury unaccompanied by marks of external violence on the surface of the body in the kidney region or anywhere else. Possibly in some children a persistence of the infantile ptosis[3] may persist leaving

[3] Aglave, Bulletin de la Soc. d'Anatomie de Paris, 1910, p. 595.

more of the surface unprotected by the thoracic bulwark. The particular vulnerability in childhood has also been ascribed to the minimum deposit of perinephric fat and the greater tension of the overlying peritoneum.

The extent of the lesion naturally runs the gamut from the mildest of superficial bruises to the complete rupture observed in my 4 cases, to the tearing away of the kidney from its vascular pedicle or the ureter or complete pulpyfying from extraordinary crushes. In the less extensive injuries it is of practical importance whether the tear involves or extends into the pelvis; whether larger vascular trunks are destroyed, with resulting dangerous hemorrhage or jeopardizing the future vitality of portions of the organ; whether the injury is subcapsular, and finally, whether there is a coexistent tear of the peritoneum or injury of the contiguous viscera. Unfortunately, few if any of these lesions can be diagnosticated with certainty as regards their extent, particularly at a period when early interference may be all important. A consideration of the nature of the violence is helpful. Injuries resulting from direct violence will probably produce a rupture of the kidney alone by "bursting" violence. Gross direct violence, such as "runover" accidents, are more likely to result in complex lesions. The intensity of the violence is, however, not a trustworthy guide, as shown by Case I, where a complete rupture resulted from the kick of a horse that left no mark on the skin. It must also be borne in mind that a pathological kidney may rupture from the most trivial accident (Watson's case of the woman whose hydronephrotic kidney ruptured from muscular action while she was washing windows).

The loss of blood resulting from any of these injuries naturally varies. Generally speaking, it is rarely sufficient to endanger life quickly; it is rather the constant and recurring hemorrhage that is most to be dreaded. Even with extensive rents of the kidney the integrity of the capsule tends by tension to check extraordinary bleeding.

As regards diagnosis, it may be stated broadly that a diagnosis of some degree of injury to the kidney presents little difficulty. Statistics give a history of hematuria in 80 per cent. of the cases, and certainly with painstaking microscopic urinary examinations this figure would be increased. It will not ordinarily be difficult to exclude lesions of other portions of the urinary tract, for example, of the bladder, practically always complicated by a fracture of the pelvis. The history or evidence of an injury which may implicate the kidney will generally be elicited; pain, tenderness, and eventually more or less pronounced signs of the extravasation of blood or urine or both in the marked cases will accentuate the diagnosis and also indicate the side involved. For unusual cases and conditions the cystoscope or ureteral catheter may be used; but as a routine these are uncalled for, as well as unwise, and in children can scarcely ever

be used, and if requiring anesthesia had better be replaced by a harmless and more satisfying exploratory and therapeutic lumbar incision.

What is most difficult is to determine the extent of the lesion, and particularly as regards the condition which most urgently call for interference. The initial symptoms with the exception of the degree of shock and hemorrhage do not present any features which sharply indicate the severity of the damage. It is rather on the development and sequence of secondary manifestation that we have to rely or perhaps waste valuable time.

Very severe injuries or very mild ones may be usually diagnosticated with readiness, especially with a definite knowledge and appreciation of the nature of the causative violence. For instance, a child is run over by a heavy wagon, as reported by a competent witness; there are extensive marks on the body; there is abundant and early and perhaps immediate hematuria; there is marked shock. Given these conditions there should be a severe laceration of the kidney and perhaps of other contiguous organs, possibly entailing a laceration of the peritoneum overlying the kidney. These complicating conditions may not always be obvious at the outset, although these marked and dangerous symptoms will manifest themselves later—too late probably to remedy them.

On the other hand, a lad may be hit a severe blow in boxing, the so-called "kidney blow." He feels a good deal of pain; may be temporarily dizzy or sick at his stomach; and sooner or later the urine is tinged with blood. Such a history and such findings indicate a trifling condition, requiring no active treatment.

It is, however, the cases of moderate severity, or of incomplete symptoms, that are the most difficult to judge. The degree of initial shock is alone no criterion; it may be intense for a short time, with only a trifling injury; it may be insignificant, or wanting, with the severest damage. The degree of hemorrhage is also misleading. A small vessel may bleed savagely for a while, and if the bulk of the hemorrhage finds a ready escape down the ureter we shall have an alarming picture for a perhaps trifling condition. On the other hand, mechanical obstacles—rupture of the pelvis or ureter (or blocking), clotting, or absence of considerable hemorrhage from the kidney—may result in little hematuria even in the presence of the severest damage.

Absence of visible marks of external violence is no criterion, for complete rupture may occur despite this negative evidence (Cases I and IV).

The significance of a swelling in the flank varies a good deal. If considerable and early it usually means extensive damage. Some of it may be due to the trauma to the abdominal wall, some to the bulk of the extravasated blood, some to the reaction of irritated intestines inhibiting peristalsis, or to an actual lesion of the gut, or

later to a peritonitis due to extravasation of urine, or an infection of the retroperitoneal tissues, or from associated injuries.

The amount of urine collecting in the tissues will depend on whether the injury involves a rupture of (1) the capsule, (2) pelvis, (3) ureter, and whether the urine can accumulate in a well-defined space, or whether opportunity is offered for extravasation into the tissues or the peritoneum. Tuffier has shown from animal experiments, and clinical observations have corroborated, that the lacerated renal surface *per se* allows little or no urine to escape.

Later swellings may be due to secondary infections. A considerable and increasing well-defined swelling (colon pushed forward) with remission of acute symptoms and absence of inflammatory signs, would indicate the retroperitoneal accumulation of a well-walled-off collection of urine which is shut off from escape down the ureter; exploratory puncture (if deemed wise) will prove the condition.

It is obvious that we are not able to diagnosticate accurately the extent of many of these lesions. We know also that many such injuries, while not rapidly producing death, may do so eventually on account of the many complications that may arise. My feeling is that we should not hesitate in dubious cases to complete our diagnosis by an early exploratory lumbar incision which will also fill a useful and probably necessary therapeutic role. Not many years ago we thought ourselves competent to differentiate the several forms of appendicitis; few surgeons today care to take such a risk, preferring to replace doubt with certainty; and I believe that the varying possibilities for harm of a kidney lesion furnish a reasonable analogy.

In regard to prognosis, statistical data of large series of cases have been collected to show results both of the condition, and the value of the various forms of treatment, but it is doubtful if the older figures have much value today.

Suter,[4] in 1905, found in a study of 701 subcutaneous injuries of the kidney: Total mortality, 18.6 per cent.; mortality of 131 treated by nephrectomy, 16.7 per cent.; mortality of 143 conservative operations, 14.6 per cent.; mortality of 427 treated expectantly, 20.6 per cent. If these figures are of any value at all, certainly an expectant treatment which has a mortality of over 20 per cent. does not make a very impressive showing. With modern technique, generalization of skilled operators, efficient means of combating shock, etc., to refrain from operation satisfied with a mortality of 20 per cent. cannot be accepted as progress. Watson showed in a series of 99 cases of operation in which the condition of the kidney called only for minor procedure there were only 7 deaths, the cause of death generally being found due to conditions independent of the operation proper (injury of the other kidney,

[4] Beit. zur klin. Chirurgie, Band xlvii.

peritonitis). Watson has formulated the indication for treatment as follows:

Cases Suitable for Expectant Treatment: (1) The milder forms of the injury. (2) The cases in which there is reason to believe that both kidneys have been injured, the signs being external evidence of injury on both sides, tumor in both loins, and anuria. (3) Cases in which there are injuries of other parts of the body of such grave character as to make futile any operative treatment of the renal lesion.

Cases Demanding Operative Treatment: (1) All in which there is evidence of progressive hemorrhage, for example, increasing pallor, pulse of declining strength and increasing rapidity, sighing respiration, and, locally, a tumor in the loin which is increasing in size, or an increasing amount of free fluid in the peritoneal cavity in the cases complicated by intra-abdominal injuries. (2) Hematuria which persists for a long time, even though the quantity of blood is at no one time large; hematuria in which there is a large amount of blood, even though it has not lasted long; hematuria which recurs after having ceased; sudden cessation of a previously profuse hematuria, and, if there is no reason to believe that both kidneys are injured, absence of hematuria. (3) Anuria which continues for more than thirty-six or, at most, forty-eight hours, and if there is no reason to believe that both kidneys are injured. (4) Cases in which there is evidence of intra- or perirenal suppuration or of peritoneal infection.

My own feeling would be that we should refrain from immediate operation in: (1) All milder cases, presenting no one symptom of any severity, and giving a history of injury which is presumably of no great violence. (2) Cases of generalized injury with a very bad general condition and absence of urgent kidney symptoms.

For the latter class I would urge an exploratory operation with an appreciable increase of any or all symptoms at an early date. Operation in some form then is indicated for all milder cases that show a tendency to increase their symptoms and for all other cases, barring those falling in Class 2. My attitude in the borderline cases would be, when in doubt, to operate, believing by such a routine measure we will not let some seemingly mild case slip through our fingers. As regards the time of operation, in general one should operate as early as possible, but if the main symptom is not that of an increasing anemia (repeated examinations of the hemoglobin), one might well occasionally give the patient a few hours in which to pull himself together, though such a delay should not be entertained if we have associated intraperitoneal injuries calling for prompt relief.

Operative Treatment. As a rule, the incision should give an extra-peritoneal approach by the lumbar route. It is the most direct, avoids infecting the peritoneum, and does not require

handling and blocking off of protruding intestines. Moreover, it will provide the safe and efficient drainage demanded in most of these conditions. An anterior incision should be reserved for injuries which presumably involve the intraperitoneal organs; even in these cases a supplementary lumbar incision for drainage may be indicated, particularly if a nephrectomy is not performed.

Treatment of the Injured Kidney. Nephrectomy should be reserved for the cases in which the integrity of the kidney cannot be preserved, and it is obvious that hemorrhage cannot be effectually stopped or prevented otherwise, or the outflow of the urine into the ureter cannot be effectually restored. In the event of doubt arising regarding the integrity of the other kidney, nephrectomy may be deferred until sufficient information is obtained. Meanwhile, the injured kidney should be attended to, the peritoneum if torn should be sutured or packed, lacerations sewed if advisable, the pelvis drained, and the whole or part of the wound packed and drained efficiently. Where nephrectomy is not required, suture or packing with drainage will suffice. How much more efficient suture rather than packing a lacerated area will prove is to me an open question. I think not much time should be lost in performing it, and it should perhaps be reserved for cases in which packing may less efficiently check bleeding. The main indication is to provide free drainage which will minimize the disastrous secondary effects of injury and extravasation.

This paper is written to call attention to the fact that rupture of the kidney in children is probably commoner than generally estimated. That the lesion is frequently severe, consisting of a complete division of the kidney into unequal halves. That shock and other symptoms may be slight and out of proportion to the gravity of the lesion. That operative interference should be more freely employed and gives good results.

THE SO-CALLED "MIXED" TUMORS OF THE SALIVARY GLANDS.

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The mixed tumors of the salivary glands were carefully reviewed by Wood in 1904. His article gives a detailed report of his observations on the pathology of 42 cases and contains a clear *resume* and

fairly complete bibliography of the pathologically significant literature to that date. His conclusions in brief are as follows:

"1. There is a group of extremely complicated tumors occurring in the facial region which contain elements from both epi- and mesoblast in most intricate relationship to each other.

"2. The complicated structure of the stroma, containing as it does elements such as embryonic connective tissue, cartilage, bone, fat, and lymphoid tissue, and very rarely striated muscle is explained most easily by the assumption of an embryonic misplacement of mesoblast.

"3. The structure of the parenchyma is so slightly characteristic in morphology that its epithelial nature in all cases can only be considered as probable; yet in about 24 per cent. of the tumors examined the presence of epithelium is undoubted. The form and relationships of the cells of the parenchyma do not furnish sufficient data to justify these cells in being regarded as of epithelial origin.

"4. The theory of early embryonic displacement of epiblastic tissue during the process of formation of the parotid and submaxillary gland and the branchial arches may account for many of the morphological peculiarities of the cells of these tumors, especially the lack of any typical features which we associate with epithelium. The same conditions may be seen in the epithelial cells of the congenital moles in which the epithelium is with difficulty distinguished from connective-tissue cells, owing to its close connection with the stroma of the tumors and its undifferentiated type.

"5. The mixed tumors of the salivary gland run a clinical course strikingly different from the sarcomas and carcinomas in that they are slow growing and generally benign. The regional lymph nodes are not invaded and recurrences are likely to remain local in a considerable proportion of the cases."

Since the publication of Wood's article an extensive literature has grown up. A bibliography thereof is appended. The following papers may be briefly noted as of pathological interest: Verhoeff reports 5 cases of tumors of the lacrymal gland and discusses the various theories of the formation of these tumors and of the mixed tumors of the salivary gland. His conclusions are as follows:

"The mixed tumors of the lacrymal and salivary gland (so-called endotheliomas) are essentially epiblastic in origin. The stroma of these tumors is not derived from mesoblastic cells misplaced from other structures, but is probably produced by an atypical development of cells which ordinarily would have gone to form part of the stroma of the normal gland. Owing to their situation the tumors of the lacrymal gland are not only dangerous to sight, but are more dangerous to life than those of the salivary gland and should be extirpated as soon as possible."

Keen observed one of the largest mixed tumors of the parotid reported, the specimen weighing seven pounds.

Ehrich reports his observations on the pathology of 33 tumors of the salivary gland. He gives a detailed report of the cases, both clinically and pathologically, and a lengthy discussion of the literature.

Martini, who has studied 6 cases, reviews the literature at great length and discusses the various hypotheses of origin of the tumors. He noted in his cases that in the peripheral parts of the tumors the stroma consisted of a more or less strongly developed framework of fibrous or reticular structure which was directly united with the capsule and that the same in advancing toward the centre of the tumors changed its structure, assuming the appearance of thick and tough or soft and loose connective tissue or a myxomatous or cartilaginous connective tissue. . . . No sharp limit existed between the various kinds of connective tissue. . . . The endotheliomatous formations were more luxuriant in those zones where the stroma had a fibrous structure. He concludes: "(1) The presence of fibrous connective tissue in the youngest parts of a tumor and of myxomatous and cartilaginous tissue in the oldest parts of the same indicate that these varieties of the connective tissue present the product of a metaplastic process of the fibrous stroma and not the product of processes developing at the cost of *germi-aberrantes* or other mesenchymal elements. (2) Besides the different phases of evolution of the neoplastic cells and their slow or rapid growth one observes the various phases of alteration and transformation of the stroma. . . . The question of the significance and origin of the myxomatous and cartilaginous tissue in the mixed tumors remains still undecided."

Martini was unable to identify the presence of true gland tissue in his mixed tumors. He asserts that the "acini" are without a limiting membrane and that the supposed epithelial cells are intimately related to the stroma. He thinks he can trace the origin of the tubules and cell formations from proliferated endothelium in the lymph vessels.

Massabuau reports on 4 mixed tumors of the parotid, 1 of the submaxillary gland and 1 of the velum palati. He discusses at length the literature and various theories of development, but finally concludes, "the theory which explains in a perfect manner the structure of these mixed tumors of the salivary gland is that which explains their development from ectomesodermic rests of the embryonic bud which was destined to the formation of the glands themselves. (1) It is the one important fact in the development of the mixed tumors on the border of glandular organs. (2) It explains the new formation of the flat epithelium as well as of the normal and edematous glandular epithelium. . . . (3) It explains perfectly the *intrication* of the two orders of endo-

thelial proliferation. . . . (4) There is no need of invoking, as in the endothelial theory, a cellular metaplasia for explaining the development of the polymorphous connective stroma of these tumors."

Speese reports his study of 9 cases, 8 of which were tumors of the parotid gland and 1 sublingual.

Fick reports a careful study of 2 cases, which he thinks do not support the hypothesis that tumors are endotheliomas.

Cordier reports in detail 1 case.

Von Hansemann has recently studied one enchondroma of the parotid in which he thinks he can without doubt trace the development of the parts from lymphangioma. He says, "I consider in fact this tumor a lucky finding which makes it possible to advance the sureness of the endothelial nature of the so-called parotid enchondromas. That lymph angiomas are present in the parotid has been long established. That, up to this time, such characteristic stages as are present in the tumor in question were not found is because these tumors were only seldom studied in the initial stages."

Sertoli makes a careful study of 4 cases and assumes that though the tumors may be of endothelial nature this is not opposed to the theory of their development from *germi aberrantes*.

The object of the present paper is to place on record the results of the analytic study of 56 mixed tumors of the salivary glands, which have been removed at operation at St. Mary's Hospital (Mayo Clinic), from January 1, 1905 to April 10, 1911.

The following tables give the sex and age incidence of onset, duration, and period of operation of the cases of "mixed" tumors of the salivary glands which we have studied:

TABLE I.—Sex and Age Incidence at Onset of Symptoms.

Decades:	2d	3d	4th	5th	6th	7th	Total.
Cases, males	3	10	10	3	3	1	30
Cases, females	6	5	6	6	3	..	26
Total	9	15	16	10	6	1	56

TABLE II.—Time from Onset of Tumor to Date of Operation.

Time:	1 mo.	4 mo.	8 mo.	9 mo.	1 yr.	2 yr.	3 yr.	4 yr.	5 yr.	6 yr.	7 yr.	8 yr.	10 yr.	13 yr.	15 yr.	20 yr.	25 yr.	27 yr.	30 yr.	44 yr.	Total.
Cases, males	1	1	1	1	4	5	1	.	1	3	1	1	3	1	2	1	.	1	1	.	30
Cases, females	1	.	.	.	5	4	3	2	2	4	1	2	.	.	.	.	1	.	.	.	26
Total	2	1	1	1	9	9	4	2	3	7	2	3	3	1	2	1	1	1	1	1	56

TABLE III.—Sex and Age Incidence at Time of Operation.

Decades:	2d	3d	4th	5th	6th	7th	8th	Total.
Cases, males	2	5	8	8	3	3	1	30
Cases, females	3	4	7	5	4	3	..	26
Total	5	9	15	13	7	6	1	56

From these tables it will be seen that the sexes are almost equally divided, but with a slight preponderance of males. From Table I above it will be seen that the age at onset in more than two-thirds of the cases is under forty years, while in one-half the cases it falls between twenty and forty years. These figures agree, on the whole, with those of other observers. For example, of Wood's 42 tumors, 4 appeared in the first decade, 1 in the second, 13 in the third, 8 in the fourth, 9 in the fifth, 4 in the sixth, and 3 in the seventh. Thus almost two-thirds of his cases also began before the fortieth year. When the figures for Wood's series and ours are added, the percentages by decades at time of onset are approximately as follows: First decade, 4 per cent; second decade, 10 per cent.; third decade, 28 per cent.; fourth decade, 24 per cent.; fifth decade, 19 per cent.; sixth decade, 10 per cent.; seventh decade, 4 per cent. These percentages are considerably advanced over those of Kaufmann and Küttner, who describe the majority of these growths as beginning in the second decade. Looked at broadly, the salient point of these figures is that 66 per cent. of the tumors began before forty years of age and 85 per cent. of them before fifty years of age.

Some interest attaches to the length of the period between the onset of the tumor and the time of surgical operation. As will be seen from Table II, this varies from one month to forty-four years, with a large preponderance of the cases running from one to ten years, and an average of six years and four months. The average of Wood's cases for the same period was eight years and nine months. When the figures for his 42 cases and our 57 cases are combined (total 99 cases) they give an average duration of symptoms prior to operation of seven years and four months. The obvious lesson from this is that these tumors do not cause sufficient inconvenience to the patients to make them seek operation early. In only 14 of our cases was there a history of pain. It is difficult to say, whether, if the patients were seen earlier, it would have a material effect on the final results of the operation, since, as will be noted later, the tumors which are carried longest without giving unpleasant symptoms are those which are apparently benign. The age at operation, as shown in Table III, indicates that the majority of these patients are in a favorable period of resistance.

Acute Parotitis. Certain observers have attempted to trace a relationship between mumps and "mixed" tumors of the salivary glands. Of the 21 of our patients with tumors of the parotid who could give a definite history concerning mumps, 14 (66 per cent.) were sure they had never had the disease. In other words, there seems to be no etiological relationship established by the statistics of mumps and "mixed" tumors of the parotid.

Location and Gross Appearance. Of our 56 tumors, 50 were in the parotid and 6 in the submaxillary glands. Thirty were on the right side and 26 were on the left side. Grossly, the tumors

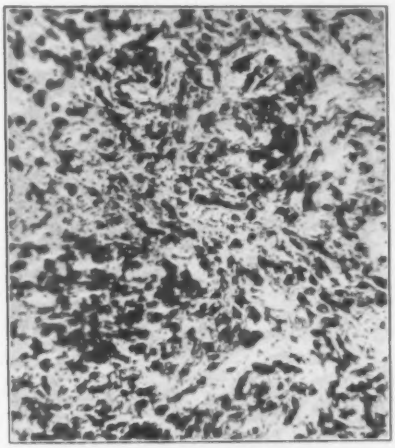

FIG. 1.—Case XIII. Group 1. Dense fibrous tumor.

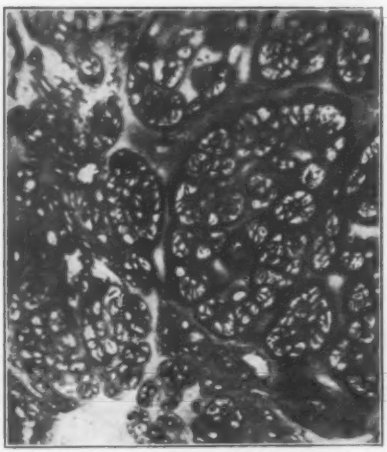

FIG. 2.—Case X. Group 2. Area showing cartilage.

may be divided into four groups: (1) Hard, fibrous masses with little cartilage and with little parenchyma (see Fig. 1); in this group were 15 cases. (2) Very hard tumors containing large cartil-

age masses (see Fig. 2); in this group were 17 cases. (3) Soft, more or less sarcoma-like tumors with relatively small amount of

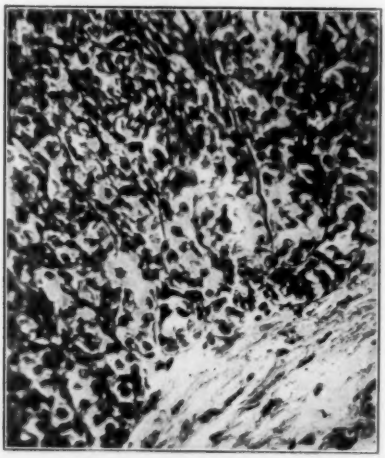

FIG. 3.—Case XVIII. Group 3. Loose parenchyma in cordons.

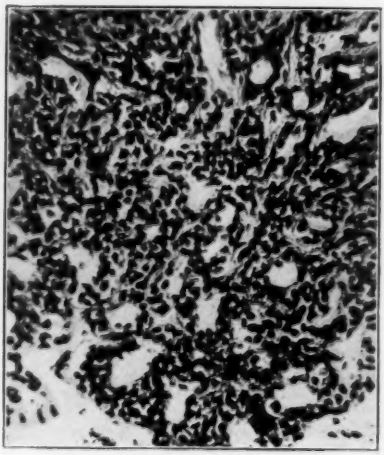

FIG. 4.—Case XXXV. Group 3. Parenchyma showing tubules.

fibrous connective tissue and a relative large amount of parenchyma cells (see Figs. 3 and 4); in this group were 20 tumors. (4) Tumors grossly resembling carcinoma; in this group were 4 tumors.

Histology; *Stroma*. Thirty-three of our cases showed marked increase of the fibrous connective tissue; 17 of these showed cartilage formation. In all of the cases in which the parenchymatous element was in marked preponderance the connective tissue appeared to be of distinct embryonic type. This connective-tissue type of embryonic cells was also present in some areas of the more fibrous tumors. In 35 of the cases a more or less distinctly myxomatous tissue was present (see Fig. 5). In many tumors was present a transparent, hyaline structure. It was noted that the cartilaginous and myxomatous elements were apt to be near the centre, that is, older portions of the tumors, rather than near the advancing borders where the simple fibrous connective tissue and parenchyma were greatly in predominence or constituted the whole of the tissue. This, of course, suggests a metaplastic origin of the cartilage, hyaline, and mucoid structures.

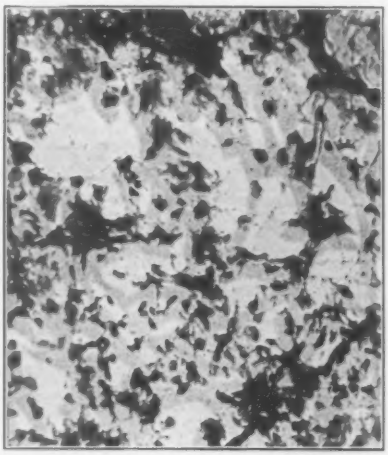

FIG. 5.—Case XVI. Group 1. Myxomatous change

Bloodvessels. In 24 cases more or less endarteritis was present. These were about equally distributed throughout the four groups as above classified from their gross appearance. In these cases the proliferation within the vessels did not seem to us as sufficient in amount or of such a type as to suggest tumor formation within them. On the contrary, the changes appeared to be rather the result of chronic irritation.

Parenchyma. In Group 1, that is, the hard, fibrous tumors, the parenchyma cells are usually found in small, distorted groups, which are frequently interpreted as the result of endothelial pro-

liferation. The same is true in certain areas in Group 2, namely, the tumors with a marked cartilaginous element. Less rarely do such cell elements occur in the tumors of Group 3, those with loose, abundant parenchyma, and then only in small areas. On the other hand, in these parenchymatous tumors the cells of the advancing neoplasms are more frequently arranged in cordons (see Fig. 3) or tubules (see Fig. 4) which strongly suggest the normal histology of the gland. A careful examination of the flattened and distorted cell groups, so frequently interpreted as "endotheliomas," leads one to the belief that they are either (1) adult epithelium which has been flattened and distorted by pressure, or (2) proliferating embryonic transitional mesothelium which has never attained

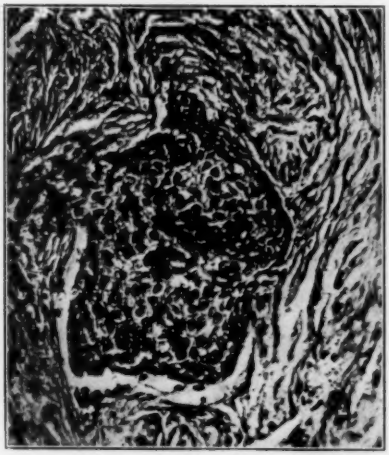

FIG. 6.—Case XV. Group 4. Mass of epithelium.

adult type. There is a direct relationship between the amount of parenchyma and the rate of development and malignancy of the tumors.

In many instances the variation in morphology in the parenchyma cells is so far retrogressive that it approaches the true spindle-celled sarcoma type. In some cases there is such a preponderance of cells of the aberrant, epithelial type (see Fig. 6) that one hesitates whether or not to diagnosticate such tumors as carcinomas. We have, however, included within our series only those cases of salivary gland tumors which show a newgrowth of two or more of the types of fibrous connective tissue, cartilage, sarcoma cells or adult epithelial cells.

All degrees of adhesion to and shrinkage away from the alveolar

walls by the lining parenchyma have been found. This point which has been made so much of by Volkmann we consider as having little diagnostic value.

Etiology. The origin and development of the so-called "mixed" tumors of the salivary glands must be considered in relation to the origin and development of other "mixed" tumors. These have recently received considerable study. Attention may be called to the articles by Stoerk and by the present writers attacking the Grawitzian hypothesis of the so-called renal "hypernephromas," and those by Ewing and Stoppato on the teratomas of the testicle.

There is, however, little to add to the discussion of the derivation of these tumors as given by Wood. Perhaps the matter of most interest related to their development is the growing belief of embryologists that the parotid gland takes its rise much earlier in the embryo than was formerly supposed. It now seems to be somewhat definitely settled that the *anlage* of the parotid appears about the fourth week instead of the eighth week of embryonic life as was formerly taught. At this period the mesoblastic tissues through which the gland bud forces itself are much less differentiated than they are in the eighth week. These facts are, therefore, in better accord with the hypothesis that the "mixed" tumors of the parotid are derived from areas of imperfectly developed mesoblastic tissue than was the former teaching of the later embryonic development of the parotid gland. So far as our personal observation goes, it would seem to us that the weight of evidence is in favor of the theory that these tumors are the result of proliferation of mesoblastic tissue perhaps displaced early in the embryonic development of the glands or which for some reason has failed to attain adult characteristics.

Results of Operation. Thirteen of our cases had been operated on elsewhere before they appeared at this clinic. These, therefore, represent cases of recurrence. Of the 42 cases which have been heard from since being operated on here, 9 have had a recurrence of the disease. Five of these were cases in which the neoplasm was of the loose, markedly sarcomatous type, and 4, cases in which the original growth was of the hard, fibrous type. Five of these 9 cases have died. Of these 5, 2 cases had tumors with a large epithelial element and in 3 the largest element of the tumors was of sarcoma type. All of these cases have died away from the clinic and no opportunities have been afforded to make postmortem examinations.

As has been pointed out by Judd, the recurrences are caused mostly by the difficulty of removing all portions of the tumor and yet preserving the fibers of the seventh nerve. When a tumor is already sarcomatous or carcinomatous at the time of operation the chances for recurrences are materially increased.

Mixed Tumors of Parotid Glands.

Group, Case No.	Registered No.	Sex.	Age.	Duration of symptoms.	Previous operation.	Date of our first operation.	Subsequent history.	Side affected.	Previous parotitis.	Pain.	Rate of tumor growth: Rapid = R, Slow = S.	Macroscopic type of tumor.	Vessels: increase = +, decrease = —; endarteritis = E.	Stroma: increase = +; decrease = —; hyalin = H.	Cartilage.	Cell groups: alveoli = A; solid masses = SM.	Cells: cuboidal = C, flat = F; spindle = S, small round = SR.	Mitotic figures.	Myxomatous changes.	Calcification.
						1905														
1	P. 2082	M	29	8 yr.	.....	Jan. 3	OK, 5 yr. 6 mo.	R	?	—	S	1	+	+H	—	—	C, S, SR	—	—	—
2	G. 4541	F	44	8 yr.	.....	Mar. 27	OK, 4 yr.	R	?	+	R	1	—	+	—	—	S	—	—	—
3	G. 4758½	M	50	1 yr.	.....	May 10	O. K., 3 yr.	L	—	—	S	1	—	+	—	—	S	—	—	—
4	P. 2691	F	37	7 yr.	.....	May 25	O. K., 5 yr.	L	+	—	S	1	—E	+H	—	SM	F, SR	+	+	—
5	J. 812½	F	44	1 yr.	.....	July 10	Rec., 5 yr.	L	?	+	S	3	—	—	—	SM	C, S	+	+	—
6	3043	M	33	2 yr.	.....	July 20	Died 1 yr. 3 mo.	L	?	+	R	3	—	—	—	SM	C, F	+	—	—
7	S. 302	F	34	1 yr.	.....	July 25	Rec., 2 yr.	L	?	+	R	3	—	—	—	SM	SR	+	+	—
						1906														
8	Mrs. J. W.	F	57	8 yr.	.....	Mar. 7	O. K., 4 yr. 3 mo.	R	?	—	S	2	—	+H	+		C, F, S	+	+	—
9	J. 1597	F	47	5 yr.	.....	June 23	O. K., 4 yr.	R	—	+	S	1	—E	+H	—	SM	F	—	+	—
10	Miss A. S.	F	20	3 mo.	.....	July 25	O. K., 4 yr.	L	—	—	S	2	—	+H	+	SM	C, F	—	+	—
						1907														
11	Pl. Bk., 14	F	38	2 yr.	.....	Mar. 18	O. K., 3 yr. 4 mo.	R	?	·	S	1	—E	+H	—	—	S	—	+	—
12	P. 6922	M	50	2 yr.	.....	April 10	O. K., 3 yr. 2 mo.	L	+	—	S	2	—E	+H	+	SM	F, S	—	+	—
13	Be., 61	F	56	7 yr.	.....	May 22	O. K., 3 yr.	R	?	—	S	1	—E	+H	—	SM	C, F, S	—	+	—
14	Gif., 1764	M	19	1 yr.	.....	May 22	Rec. 1 yr. 6 mo.	R	?	—	S	3	+	—	—	SM	C, S	+	—	—
15	J. 2673	M	28	1 yr.	.....	May 28	Rec. 3 yr. 4 mo.	L	?	—	S	3(4)	—	+H	—	SM	C, F	+	+	—
16	Dr. A. M. J.	M	37	2 yr.	.....	June 10	O. K., 3 yr.	L	—	—	S	1	—	+H	—	—	S	—	+	—
17	Bk. or M6	F	39	2 yr.	.....	June 11	.....	L	?	—	S	2	—E	+H	+	—	C, F	—	+	—
18	J. 2738	M	47	10 yr.	9 yr.	June 15	O. K., 3 yr.	L	+	+	S	3	+	+H	—	A, SM	C, F, S	+	+	—
19	J. 2830	M	31	12 yr.	.....	July 15	Rec., 3 yr.	R	+	+	S	3	+	+H	—	SM	C, F	—	+	—
20	Mr. C. J. F.	M	37	10 yr.	.....	Aug. 7	O. K., 3 yr.	L	±	—	S	2	+E	+H	+	A	C, F	—	+	—
21	A1712	M	70	30 yr.	Inf.	Sept. 24	Rec., 6 wk.	R	?	+	S	4	—E	+H	—	—	C, F	—	—	—
											3 mo. R									
22	A3942	F	20	1 yr.	.....	Nov. 23	.....	L	?	—	R	3	—E	—	—	A, SM	C, F	+	—	—
						1908														
23	A5387	F	25	1 yr.	.....	Jan. 11	O. K., 2 yr.	L	+	+	S	1	—E	+H	—	SM	C, F	—	—	—
24	A7340	F	27	5 yr.	Op., 3 yr. 6 mo. Rec., 1 yr. 6 mo.	Mar. 7	Rec., 2 yr.	R	?	+	R	1	—	+	—	—	F, S	—	+	—
25	A11599	M	43	15 yr.	14 yr.	July 22	O. K., 2 yr.	R	?	+	S	2	+E	+H	+	SM	C, F	+	+	+
26	A18330?	F	18	6 yr.	.....	Sept. 10	O. K., 2 yr.	R	—	—	S	3	—	—	—	A, SM	C, S, SR	+	+	—

27	A12357	F	34	2 yr.	.....	July 27	?	L	—	+	S	2	—E	+H	+	SM	F, S	—	+	+
28	A5175	F	31	6 yr.	.....	Jan. 6	Rec., 1 and 2 yr.	L	—	+	R	3	—E	+H	—	A, SM	C, F	+	+	—
29	A18358	M	61	4 mo.	.....	Dec. 28	Died 1 mo. later	L	?	+	R	4	—	—	—	SM	F, C	+	+	—
						1909														
30	A4689	M	38	7 yr.	.....	Feb. 6	O. K., 1 yr. 6 mo.	L	?	—	S	2	—E	+H	+	A, SM	C, F	+	+	+
31	A22140	F	50	7 yr.	.....	April 6	O. K., 1 yr. 2 mo.	R	+	+	S	1	—E	+H	—	A	SR	+	+	—
32	A21697	F	34	8 yr.	Op. 1 yr. ago	Mar. 27	Rec., 1 yr.	L	?	+	S	3	—	+H	—	SM	F, C	+	—	—
33	A22675	F	62	2½ yr.	.....	April 21	O. K., 3 yr. 4 mo.	R	..	..	S	2	—E	+H	+	A, SM	F, S, SR	—	+	—
				6 mo.																
34	A26098	F	24	6 yr.	Op., 3 yr.	Oct. 14	Rec., 6 yr.	L	?	+	R	3	+	—	—	SM	F, C	+	—	—
					Rec., 6 mo.		Died 1 yr. 2 mo.													
35	A31134	F	43	3 yr.	.....	Nov. 13	O. K., 1 yr.	R	?	+	S	3	—E	+H	—	SM	F, S	+	—	—
						1910														
36	A33898	M	27	3 yr.	.....	Feb. 9	O. K., 4 mo.	R	?	+	S	3	—	+H	—	SM	F, C	+	—	—
37	A34574	M	30	6 yr.	.....	Feb. 25	O. K., 4 mo.	R	?	—	S	2	+E	+H	+	A, SM	C	+	+	—
38	A37071	F	54	4½ yr.	.....	April 3	O. K., 2 mo.	L	?	—	S	3	+	+H	—	SM	F, S	+	+	—
				6 mo.																
39	A37735	M	71	1 yr.	10 mo. ago	June 19	Died 3 mo.	L	?	+	R	3	—	—	—	SM	S	+	—	—
					25 yr.		Rec.													
40	A38746	F	62	25 yr.	20 yr.	June 14	O. K., 3 mo.	R	—	+	R	2	—	+H	+	A, SM	F	—	+	+
					2 yr.															
41	A39230	M	55	20 yr.	.....	June 23	.....	R	?	—	R	3	—	—	—	A, SM	F	+	—	—
42	A39803	M	57	27 yr.	27 yr.	July 8	.....	L	?	+	S	3	—	—	—	A, SM	C, F	+	+	—
					17 yr.															
43	A40330	M	25	5 yr.	.....	July 16	Rec. after 4 mo.	R	—	+	S	3	+	—	—	A, SM	C, F	+	+	—
44	A14953	M	39	2 yr.	.....	Aug. 22	.....	R	?	+	S	2	—	+H	+	A	C, SR	+	+	—
45	A42737	M	42	2 yr.	.....	Sept. 2	.....	L	?	—	S	2	—	+H	+	A	F, C	—	+	—
46	A28505	F	58	1 yr.	.....	Oct. 1	.....	R	?	—	S	2	—	+H	+	A	C, F	—	+	—
47	A44948	M	60	8 mo.	6 mo. ago	Oct. 25	.....	L	?	—	R	3	+	—	—	A, SM	C, F, SR	+	+	—
48	A46670	M	41	13 yr.	2 rec.	Dec. 12	.....	R	?	—	R	1	—	+H	—	A	C, SR	+	+	—
						1911														
49	A48731	F	24	4 yr.	.....	Feb. 7	.....	R	?	—	S	1	—	—	—	—	C, SR	—	+	—
50	A51433	F	64	44 yr.	.....	April 6	.....	R	?	—	S	4	—	—	—	SM	F, C	+	+	—

Mixed Tumors of Submaxillary Glands.

						1905														
1	P. 2463	M	39	6 yr.	.....	Apr. 8	O. K., 5 yr. 2 mo.	L	?	—	S	2	—E	+H	+	SM	C	+	+	—
2	G. 5570	F	46	1 mo.	.....	Oct. 16	O. K., 4 yr. 8 mo.	R	—	+	R	4	—	+	—	A, SM	C, F, S	—	+	—
						1906														
3	J. 1255	M	61	9 mo.	.....	Mar. 27	O. K., 4 yr.	L	—	—	R	1	+	+	—	—	S, SR	—	+	—
4	M. 2552	M	16	6 yr.	.....	June 5	.....	R and L	—	—	S	2	—E	+H	+	SM	C, S, SR	—	+	+
						1909														
5	A21949	M	49	15 yr.	.....	Mar. 31	.....	R	—	+	S	2	—E	+H	+	SM	C, S	+	+	+
6	A31575	M	40	1 mo.	.....	Dec. 1	.....	R	+	+	R	1	+E	+	—	—	S	—	+	—

Summary. 1. The present study covers 50 "mixed" tumors of the parotid and 6 "mixed" tumors of the submaxillary glands.

2. Most of the "mixed" tumors of the salivary glands arise in young adults.

3. Most of the tumors cause little inconvenience early and surgical relief is not sought until an average period of about seven years has passed.

4. Our cases show no etiological relationship between the tumors and acute parotitis.

5. The tumors which grossly are hard, consisting of fibrous connective tissue or cartilage are proliferating at their periphery only and are usually benign.

6. The tumors which grossly are soft, consisting largely of parenchyma of types varying from adult to embryonic are those among which are found the malignant tumors.

7. Connective-tissue and parenchymatous elements both tend to revert to embryonic type.

8. There is little evidence that these tumors arise from proliferating adult epithelium, or endothelium.

9. There is considerable evidence to support the theory that these tumors are mesotheliomas of embryonic origin.

10. After fairly complete operative removal few of the tumors recur.

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DUODENAL ALIMENTATION.[1]

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When Einhorn first described his method of duodenal alimentation about a year and a half ago, I realized its possibilities and began using it in certain selected cases. After a considerable

[1] Read before the New York Academy of Medicine, February 15, 1912.

experience I regard duodenal feeding as a distinct mark of progress in the treatment of those conditions where rest for the stomach is indicated. The principal field for its usefulness lies, of course, in the treatment of certain gastric and duodenal ulcers, but its scope can be legitimately enlarged by including certain other conditions, as will be shown later.

For those who are not acquainted with the duodenal tube, I will briefly outline the technique of its use. The tube is swallowed by the patient at bedtime, with the aid of water. When the first mark on the tube is at the teeth the bucket on the end is entering the stomach. The second mark indicates the distance from the mouth to the pylorus. During the night the capsule, provided there is no obstruction, will usually have passed the pylorus and the third mark on the tube should be at the teeth, indicating that the end of the tube is *in situ*, well down the duodenum.

Before starting the feeding it is well to make sure that the tube is really *in situ*. This is done in several ways. Gentle traction will develop a slight resistance if the tube is in the duodenum. Aspiration through the tube will often recover golden-yellow duodenal juice and not gastric secretion. Lastly, and most certain, if the patient be given some liquid to drink by mouth and aspiration is immediately performed it will not be returned through the tube as would be the case if the latter were coiled up in the stomach.

When certain that the tube is in place we can proceed with the feeding. The food which has been found most satisfactory is a mixture of milk, sugar of milk, and raw egg. The amount given at a feeding should be small at first, 100 c.c. every two hours from 7 A.M. to 9 P.M., increased until 280 or 300 c.c. are taken at each feeding, if possible. When the patient is taking 280 c.c. of milk, one egg, and one tablespoonful of sugar of milk at each feeding he is receiving approximately 2840 calories for the total eight feedings in the twenty-four hours. As the average individual while in bed requires under 2000 calories to maintain his nitrogenous equilibrium, it can be seen that there is a liberal amount in excess for the purpose of building up the patient.

Einhorn uses the glass syringe to administer the food, and by means of a feeding table which fits over the glass of nourishment, is able to fill the syringe from the glass and by a manipulation of petcocks force the contents into the duodenal tube without disconnecting.

In treating my first 2 patients I thought it would be advantageous to employ a method like that of Murphy for giving salt solution per rectum, and so I have made use of gravity from a pint irrigating jar, connecting its tube to the duodenal tube by a glass connection and setting the rubber petcock so that the nourishment enters the duodenum slowly, about twenty-five minutes being required for the larger feedings.

Duodenal alimentation is not, as a rule, suitable for use in the home unless a nurse who has had some experience with it is in constant attendance. There are a number of points which must be adhered to rigidly or untoward happenings will occur. As a rule, if the patient's relatives are entrusted with giving the feedings some mistake will result in the patient being made uncomfortable or the tube being blocked up so as to necessitate its withdrawal in order to clean it.

The food must be administered at body temperature. Food normally entering the duodenum from the stomach is at body temperature, and so the former is not accustomed to shocks of too high or too low temperature, and resents such insults by distressing symptoms of shock, cold perspiration, flatulence, and distress, and perhaps even syncope. The mixture should be heated gradually, preferably in water, and should not be made too hot lest it become thick and lumpy from cooking of the egg. After heating it should be strained. This will help in preventing the tube from becoming occluded.

The food should be allowed to enter the duodenum slowly. Too rapid administration will cause flatulence and often the same symptoms as variations in temperature.

At the termination of each feeding a syringeful of water at body temperature should be injected; then the petcock closed; the syringe filled with air; the petcock again opened and the air injected, after which the petcock should again be closed before the syringe is disconnected. This keeps the tube cleared and clean. The management of the petcock is not trivial, as it might seem, and if, say, the petcock was not turned off when the syringe is removed, some of the food would likely rush back into the bucket and lower tube and be converted by the trypsin into a cheesy lump, defying removal until the tube is removed and cleaned. It is because of the importance of these details that one who has had experience should superintend the feedings.

Duodenal feeding is only to be chosen for certain cases. Certain ulcers with severe symptoms, which would heretofore have been treated at the beginning by rectal feeding for some days, may at once be put upon duodenal feeding. Again, in an ulcer which has not healed under a thorough course of medical treatment we should try duodenal alimentation before resorting to surgery. By so doing we can eliminate several of the factors which tend to prevent ulcer from healing, that is, the presence of acid in the stomach, irritation of food in the stomach, stretching of the stomach, and peristalsis. At the same time the body secures sufficient nourishment.

This method of treatment will not cure all cases of ulcer, and some may have to be operated upon later; but in all in whom I have tried it, the general condition of the patients has been decidedly

improved, so as to render them better surgical risks. The following synopses of 2 cases which later came to operation show this point.

Miss M. T. D., aged thirty-four years, had symptoms for nine years, complaining of pain and tenderness in the left epigastrium, coming on from two to three hours after meals, and severe headaches. The pain was decidedly aggravated whenever she was much on her feet. There was no nausea or vomiting. Her gall-bladder was distinctly palpable and slightly tender, but not distended. The right kidney was palpable to the second degree. A spot of some tenderness was found on deep pressure, one-half inch below and one inch to the left of the ensiform. Splashing sound extended over the entire epigastrium to the navel. There was slight hypersecretion and an acidity of 64. The diagnosis was not clear for several weeks, as occult blood was absent from the stools, but this appeared later, at which time the thread test also became positive. After she had been on the rest treatment for two weeks and her symptoms were worse, if anything, it was thought best, in consideration of the probable long duration of the ulcer, to put her on duodenal feeding at once. She improved greatly on the treatment, but after a time her symptoms reappeared and a gastro-enterostomy was performed, an ulcer being found in both the stomach and duodenum.

Another case, that of Mrs. M., is interesting, as at operation later on, scars of two healed ulcers were discovered demonstrating that the duodenal feeding had been successful, but that other ulcers had developed after a year of health. She came to me with symptoms of recurrence of an ulcer, which had apparently been healed by medical treatment the year before. Her weight was only ninety-eight pounds. She complained of a severe epigastric pain and tenderness. Occult blood was present in considerable amounts and the thread test was positive. Operation was thought advisable, but it was decided to first try duodenal alimentation, as her general condition was very poor. The tube was left *in situ* for eighteen days, during which period her symptoms entirely disappeared. Her weight progressively improved, and in several months it reached 116 pounds, a gain of 18 pounds. Later the symptoms of ulcer recurred, and Dr. Howard A. Kelly found at operation several scars in the stomach from the ulcers healed by the former treatments and an open duodenal ulcer which was producing the symptoms at the time.

Ulcers at the pylorus, producing obstruction, may be treated with duodenal alimentation, provided the duodenal tube can be passed into the gut; but if the pylorospasm persists they then demand gastro-enterostomy. Duodenal alimentation or other forms of medical treatment should not be given too long a trial in an unhealing ulcer at the pylorus, even if not producing obstruction, because of the danger of malignancy. But in other ulcers this method of

treatment should be instituted in nearly all cases after ordinary medical means have failed to produce a cure, and will often bring about happy results. I have never had a complete failure in any ulcer case in which I have used the tube, that is, one in which no improvement whatever was noted. Many complete cures have resulted, as far as can be determined, and the remainder were greatly improved.

The cases which were not of long duration have almost without exception done well, as in the following:

Mrs. H. C. M. had symptoms of two months' duration when she first consulted me. She complained of a burning pain in the stomach and pyrosis about three hours after meals, and in the early morning, relieved for a time by food, soda, or belching. Ulcer was not diagnosticated at first since examination failed to show tender spots, and the stool was negative for occult blood. The right kidney was palpable to the first degree and splashing sound was obtained over the whole upper abdomen as far down as the lower border of the navel. Free acid was 34 and total acid 48. Under a general reconstructive treatment she at first improved somewhat, but became decidedly worse while on her vacation, so that three months after her first examination she returned with exaggerated symptoms and a positive thread test. Duodenal feeding resulted in complete relief from her symptoms, and a negative thread test, which has persisted.

Mrs. O. came to me in the spring of 1911, shortly before I left town on my vacation. Her family history was good. She had been delicate as a child, and had always been troubled more or less with digestive disturbances. During the previous two years she had been having attacks of sharp pain in her stomach, which at first would come on several hours after her evening meals. She also complained of distress in the region of the sigmoid, more or less constant in the earlier hours of the day, which to a great extent passed off after bowel actions. There was a decided tendency to a relaxed condition of her bowels. She was troubled with a continuous dull headache. Chest organs were normal. Her abdominal walls were relaxed. Splashing sound of stomach reached from ensiform to one inch below navel. A good deal of tenderness was elicited by pressure over her sigmoid, but at no other point. Her hemoglobin was 75 per cent.; leukocytes, 10,400, with a polymorphonuclear count of 75 per cent. The urine showed a very faint trace of albumin. The feces showed a good digestion and no occult blood. Stomach digestion was good, free HCl being present with an acidity of 56 and no occult blood. Because of a tendency to diarrhea, the distress in her sigmoid in the early part of the day, the tenderness to palpation in that region, the blood picture, and her progressive loss of strength and some weight, I feared lest a beginning malignant change was occurring. However, as

she and I were just leaving town for the summer nothing further could be done other than to regulate her diet, etc. In the early fall upon my return she again came to see me, and I found her general condition unchanged. Her chloroanemia was slightly more pronounced but the leukocytes and polymorphonuclear count were both quite normal. There was less sigmoid distress although the bowels were acting from three to eight times each day, the feces being always loose and mushy. However, the epigastric pains were now more pronounced and occurred from one to three hours after every meal, being worse after the heavier and more complex meals. Her bowels began to act from one-half to two hours after every meal, remarkably coincident with the onset of her pains. A point of marked tenderness was elicited below the right costal border, the thread test was positive, and occult blood was present when on a meat-free diet. She was put upon the conventional rest treatment for cure of ulcer, and kept there for several weeks, but without any marked amelioration in her symptoms for any length of time. Every attempt to increase her diet brought all her former symptoms. At the end of this time I put her upon duodenal feedings in the manner described in the previous cases. She kept the duodenal tube *in situ* for thirty-two days continuously without having it removed once, and without the slightest untoward symptoms. From the end of the first forty-eight hours she was free from all her subjective symptoms. At the end of the third week her bowel actions were reduced to one normal stool daily, and at the end of the fourth week the feces were free from occult blood. The tenderness had disappeared from the epigastrium, although there was still a sensitive area over the sigmoid. At the present time she has been on nearly a normal diet and has been about the house for some weeks; her blood is normal, and she has regained her normal weight and strength.

Such cases as these I could multiply many times over in my experience to illustrate the great reliability and usefulness of duodenal feeding in similar cases.

Einhorn in some cases of gastric ulcer, recommends the local application of healing agents by painting argyrol in nearly insoluble form on the tube at the point where it rests against the ulcer. I have not had a large experience with this method, but I have administered various drugs through the tube for other conditions arising during the course of duodenal feeding. In the diarrheas which so often accompany duodenal ulcer, prompt relief is often secured by 1 grain of argyrol in 1 ounce of warm starch water given through the tube. Flatulence caused by the milk not agreeing perfectly can often be controlled by the addition of 30 drops of glycerin to the feeding. If flatulence still persists, 5 drops of ichthyol in a little water through the tube is generally followed

by good results. Where the stool shows deficiency of bile we have often seen the yellow bile color reappear promptly upon the administration of 1 or 2 minims of dilute hydrochloric acid in 1 ounce of water through the duodenal tube.

Apart from the treatment of ulcers, duodenal alimentation may be employed with benefit in some other cases. I have secured happy results in the treatment of duodenal catarrh by using a long duodenal tube. Warm Carlsbad water can also be administered through the tube at the same time.

In one case of cancer of the stomach in which there was marked dysphagia and a sensation of closing up of the throat, so that partial starvation was resulting from the patients failing to take sufficient nourishment, I was, after two attempts, able to get the tube to pass the pylorus, and the patient was afforded considerable relief, and his strength improved for a time, by feeding through it.

There is another class of cases in which results have been most striking—namely, cases of visceroptosis of marked degree, with ischochymia, where it seems as if the stomach is incapable of emptying itself and passing along sufficient of the forced feeding to increase weight and restore the general tone. This to my mind is a strong indication for duodenal alimentation.

The stomach is given a rest, and at the same time food can be introduced into the body in sufficient quantities to cause increase in weight and improvement in muscle tone. A striking example of this is the case of Mrs. D. T. D., who was emaciated, and had a marked visceroptosis, with ischochymia. The lower border of her stomach was three fingers' breadth below the navel. She took the tube for twenty-one days and again for fifteen days. She did well from the start, and the gain was spectacular. Altogether she has gained 34 pounds, and it seems to be permanent. Previous to the duodenal feeding she had made but little progress.

Miss R. A. had marked enteroptosis for seven years, which had been treated without benefit by many physicians. She was much emaciated and a pitiable object. A long rest cure with forced feeding helped but little, but after a course of duodenal feeding she has made marked progress, and has gained 40 pounds.

I consider that duodenal feeding constitutes a mark of great progress in medicine, filling a long-felt want, a method of resting the stomach, and at the same time affording the economy abundant nourishment. In those cases in which it is indicated the results are most gratifying.

TYPHOIDAL HEMIPLEGIA WITH REPORT OF THREE CLINICAL CASES AND ONE WITH NECROPSY.

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Hemiplegia following or occurring during typhoid fever is a comparatively rare complication, and any cases of this character are well worth reporting. It has been mentioned as early as 1860 by Gubler,[1] but since then, to my knowledge, there have been few cases published.

Among the more recent literature on this subject the paper of Smithies[2] takes up and classifies the symptoms, prevalence, age, time of occurrence, etc., in considerable detail.

In compiling the cases already published, with those described in this paper, several points are of interest.

The male sex was more often affected (70 per cent.) than the female (30 per cent.) in the 40 cases in which the sex was defined.

Where the side involved was tabulated, left hemiplegia occurred in 19 (44 per cent.) and right hemiplegia in 24 (56 per cent.).

In 28 cases of right side involvement aphasia was noticed in 23 (82 per cent.), while it is mentioned in only 3 (18 per cent.) of the 17 left hemiplegics.

The complication occurred before the age of ten years in 9 instances (24 per cent.), before twenty in 10 cases (26 per cent.), and in 19 (50 per cent.) before the age of thirty years.

Of those cases in which the time of onset was given, 10 (24 per cent.) occurred in the first and second week, 8 (20 per cent.) during the third week, 4 (10 per cent.) during the fourth, 15 (40 per cent.) during convalescence, and 2 (5 per cent.) so late after the attack as to render the dependence of the condition upon typhoid rather doubtful.

Athetosis was reported by Curschmann,[3] Osler,[4] and Barrett,[5] and is also present in my first case.

Visual defects have not occurred, with the exception of a left-sided amaurosis and nerve atrophy in an interesting case described by Benedikt.[6] Here there was also stuttering with the left-sided hemiplegia. Stuttering was also one of the symptoms of my first case. In Pigott's[7] case the pupils were dilated, the right larger than the left and less reactive to light than the left,

[1] Arch. Gén. d. Méd., 1860.

[2] Jour. Amer. Med. Assoc., August 3, 1907, vol. xlix, No. 5. In this paper a very complete list of literature up to 1907 is given.

[3] Nothnagel's Handbuch, Band iii, I Teil.

[4] Johns Hopkins Hosp. Rep., 1899 to 1900.

[5] Report of Michigan State Asylum for Insane.

[6] Elektrotherapie, 1868.

[7] Maryland Med. Jour., 1909, lii.

also deviation of eyes to left. This was a left hemiplegia. A case of left hemiplegia reported by Parisot,[8] which ran a course of a few days, showed inequality of the pupils and strabismus. In Case IV there was deviation of the eyes toward the right with left-sided hemiplegia.

Sensory changes in most instances were absent or so slight as to be doubtful in value. Such changes were present in the case of Raschofsky's[9] and Case I of this paper.

An interesting case, more of a meningitic type, is described by Laure[10] in which, in addition to the typical signs of a meningitis, there occurred an aphasia with loss of ability to write, but no paralysis.

The hemiplegia occurring in typhoid does not differ materially from one following any other cause, and should therefore be readily diagnosticated. In some cases the onset may be acute, with convulsions or delirium or a rapidly developing coma or general restlessness with increase in the temperature, while in others it may be very gradual, extending over a period of hours or days or even weeks (Nothnagel), and in some instances reported there was no positiveness concerning the exact time of the occurrence of the paralysis.

This variety of onset is dependent to some extent upon the reactive or resistant condition of the patients' nervous system, but much more upon the character of the pathological lesion causing the condition. Thus, in the early cases where there is perhaps more likely to be a hemorrhage or an embolus, the onset is acute and there may be convulsions and coma. When, however, the symptoms are the result of a toxemia, there would more probably be a delirious condition of some duration, with temperature elevation in cases of an encephalitic character. In the convalescent stage there would be, more likely, a gradual onset, such as might occur in thrombosis, this lesion being in all probability the one which most frequently occurs during this stage of the disease.

A higher percentage of cases has been reported as occurring during the stage of convalescence, and this is due in all probability to the generally weakened condition of the patient at this period, the sluggishness of the circulation, and the condition of the vessels and the blood, tending to aid in the formation of thrombi in the cerebral vessels just as they do in the better known thrombotic processes elsewhere in the body.

The prevalence of this complication in the male sex is dependent upon a greater percentage of typhoid occurring in males, and also upon the fact that the male, through excessive venery and abuse of alcohol and tobacco, is more subject to such vascular changes as predispose to a thrombotic process.

[8] Rev. méd. de l'Est. Nancy, 1909, xli.
[9] Wien. med. Woch. 1911, No. 39
[10] Rev. Neur., Paris, 1908, xvi.

The question of athetosis and choreic movements present in Case I, also reported by Curschmann, Osler, and Barrett, is of considerable interest as it is caused by involvement of some vessel other than the one so frequently affected. It might be due to an affection of the meningeal vessels, causing irritation, or of the vessel or vessels supplying the lenticular and thalamic regions.

The pathological condition most frequently occurring in these hemiplegics is in all probability a thrombosis, this being due in great part to the causes mentioned earlier in my paper. Thrombi were found in the Sylvian fissure branch of the middle cerebral artery by Dankin,[11] Hawkins,[12] Osler,[13] Barrett,[14] and in my fourth case. Embolic softening would in many cases be difficult to distinguish from thrombosis—cases of this nature were published by Curschmann[15] and Hruska.[16]

Parisot[17] reported a case in which the internal carotid was thrombosed at its entrance to the skull, and as a result the central part of the right hemisphere, the external capsule, and basal ganglion were totally destroyed, leaving only a shell of the cortex intact. Curschmann's case had a diffuse hemorrhage in the lenticular area.

Some of the hemiplegias, particularly the more transient and rapidly healing ones, may be caused by a toxemia affecting the cerebral cells, and this interpretation would more readily explain the great percentage of cases having a complete or almost complete recovery. Barié and Lian[18] referred to the probability of such an origin in their case.

Encephalitis may also be the basis of some of the cases, as in the one reported by Raschofsky.[19] Here the increase of the temperature at the time of the onset of the symptoms with restlessness and the recedence of the fever with the return to normal, led the author to consider an encephalitis as the cause.

The prognosis is dependent to a great degree upon the character of the lesion present. In those cases of toxemic or encephalitic origin the recovery may be almost complete. Where a hemorrhage or thrombosis is the basis of the condition, the change would be much slower and less complete. As a rule, the leg regains its power and function much earlier than the arm and hand. Aphasia, if present, may improve, but very seldom clears up entirely (Smithies). Treatment is largely symptomatic with the necessity of avoiding the use of any drugs or measures that might lead to hemorrhage. The late stages require the usual massage with general tonic treatments.

[11] Lancet, April 23, 1892.
[12] Clin. Soc. Trans., 1889.
[13] Loc. cit.
[14] Loc. cit.
[15] Loc. cit.
[16] Präg. med. Woch., 1901, Nos. 41 and 42.
[17] Loc. cit.
[18] Bull. et. Mem. de la Soc. d. Hôp. de Paris, 1907.
[19] Loc. cit.

Papers have been written on cerebral and meningeal complications of typhoid by Du Castel,[20] Cabanes,[21] Barthelemy et Durbin,[22] Starkiewicz,[23] Diller,[24] Antony and Dofter,[25] and Laignell-Levastine.[26] Cases have been reported by Lesieur,[27] and Tondi,[28] also an atypical case of post-typhoid thrombosis by Pasteur.[29] I regret that I have been unable to obtain the records of any of these.

I am greatly indebted to Dr. William G. Spiller for the privilege of reporting the following cases, 2 of which were examined by me at the University and Philadelphia Hospitals, and a microscopical examination made in the third.

CASE I.—E. K., aged twenty-two years. Admitted to the Philadelphia Hospital December 2, 1908.

Family history negative as regards possible affections of the nervous system.

Patient was well up to the age of six, when he developed typhoid fever. During the attack of typhoid fever he was confined to bed for three weeks. He remembers that he was widlly delirious during this illness, and wanted to jump out of the window. He also claims to have been unconscious during part of his illness, but does not know the duration of the unconsciousness. About two months after onset, and a week or so after beginning to improve, upon attempting to get up one morning he found that he could not use his left leg and arm, but does not know whether or not he could move his arm and leg while in bed. He was not able to walk for some time. His speech was affected so that people said he talked "tongue-tied," and he had trouble in swallowing. There were at no time any other symptoms of head involvement such as headache, vomiting, etc.

The athetoid movements of toes and fingers were present as long as he can remember, but he does not think they anteceded the attack. His left foot felt heavier and colder than the right, and he dragged it in walking.

Examination upon admission. Left arm was flexed and adducted, and was continuously and rhythmically flexed and extended. Fingers were flexed and athetoid movements were constant. The biceps was markedly contracted and prominent, and various muscles of the forearm could be seen contracting rhythmically. The left arm with a great deal of exertion could be raised to the level of the shoulder. The forearm could be flexed and extended slightly, and with very little strength. Pronation and supination were not possible. The handgrasp was very weak, as were the

[20] Gaz. des Hôp. de Paris, 1910, lxxxiii.
[21] Bull. med. del.Algerici, 1908, xix.
[22] Gaz. med. de Nantes, 1906, xxiv.
[23] Gaz. lek Warszawa, 1906, xxiv.
[24] Jour. Amer. Med. Assoc., May 27, 1905.
[25] Arch. d. méd. et pharm. Paris, 1905, xlvi.
[26] Bull. et mem. des Hôp. de Paris, 1907, xxiv, No. 30.
[27] Bull. de.la Soc. méd. des Hôp. de Lyons, 1907, vi.
[28] Napoli, 1909, xxiv.
[29] Arch. Middlesex Hospital, London, 1909, xvi.

individual finger movements. The entire upper extremity was very spastic and resistant to passive movements; no atrophy. The left lower extremity showed retained power of flexing the leg on the abdomen, but this was greatly weaker than on the right. The leg could be flexed and extended at the knee, but no movement was possible with the toes, nor could the foot be elevated. The left foot was held in a partially flexed and adducted position, and continuous athetoid movements of the toes were present. Reflexes were markedly increased with ankle clonus and a Babinski strongly positive. Spasticity with resistance to passive movement was excessive. The leg was dragged in a spastic manner, but not swung in a circle, as it so often is in hemiplegics. Hypalgesia and hypesthesia were found over the entire left side, but no change in the temperature and position senses. Some slight asteriognosis was present in the left hand. There was no involvement of rectum and bladder.

Examination March, 1910. There remains distinct foot drop and atrophy of tibialis anticus and the peroneal group. The calf muscles are contracted. The thigh muscles are still somewhat weaker than the right. At present the athetoid movements occur in the fingers and in the arm when the patient attempts to resist passive movements. There is some slight impairment of the lower facial movements.

Case II.—W. P. (No. 4218). Aged sixteen years. Male. Admitted to the University of Pennsylvania Dispensary December 11, 1905.

The patient was perfectly well up to four years ago, at which time he had a severe attack of typhoid. During this illness he had a left-sided hemiplegia, this having occurred while he was still in bed and running a temperature. The leg and arm were both affected; he could not move his fingers properly, nor the left side of the face. The patient said he could not talk for two days, although this might have been due solely to the involvement of the tongue and face. The eyes and ears seemed to be all right.

Examination on admission showed the left arm somewhat contractured and atrophied, with the reflexes increased, and the hands cyanotic and cold. No sensory changes were present. The power in the shoulder muscles was fair, but impaired. The patient had a distinctly spastic hemiplegic gait. All reflexes were increased with positive Babinski and some atrophy of the entire leg. There were no sensory changes.

Case III.—Mrs. L. G., aged thirty-two years, white. Admitted to the University Dispensary September, 1910.

The patient always had good health until four years ago (1906), when she had typhoid fever, and was in bed twenty-one weeks. During her illness her right arm and right leg would frequently drop over the side of the bed and would have to be lifted back.

They were manipulated by the nurse, and when handled were always stiff and sore. When the patient got out of bed she experienced no especial difficulty in walking or standing, although she had to use a cane. The arm and hand were used only with great difficulty, and their original strength has never returned.

The right upper limb has never recovered in strength or size. She uses it awkwardly, and is forced to make more fine movements with the left hand. When she walks the right foot is dragged along the ground.

Examination. At rest the right side of the face shows some drooping in the lower half. Actively this is less marked, but still present. She winks the right eye less than left; wrinkles brow well. The tongue deviates to the right on protrusion. Ocular movements are good; reflexes normal. The upper extremities show marked weakness in all movements of the right arm, with all reflexes considerably increased. The entire limb is smaller than the left. There are fibrillary twitchings, but no sensory anomalies. The left arm is normal. There is no vesical or rectal disorder.

Lower extremities. Gait is unilaterally spastic. The patient drags the right foot. Individual movements are fairly well performed with right leg, very well with left. The whole right lower limb is distinctly smaller than the left. There are no fibrillary twitchings or sensory changes.

Reflexes. On the right the patellar tendon and Achilles tendon reflexes are greatly exaggerated. Ankle clonus is not demonstrable, probably on account of spastic contracture of Achilles tendon. The Babinski sign is present. On the left the reflexes are somewhat increased but not abnormal, and no Babinski.

Other than a slight thickness of speech during the first period of the attack, there was no disturbance of speech.

Case IV.—B. C., male, colored, aged twenty-four years. Admitted to Dr. B. F. Stahl's ward in the Philadelphia General Hospital (and examined by Dr. Spiller), January 6, 1906. Died January 10, 1906.

During the course of typhoid fever the patient sat up in bed and fell out toward the right, striking first upon his buttocks. He was put back to bed and Dr. Spiller, upon examining him, found a left-sided hemiplegia with deviation of the eyes away from the paralyzed side. The fall was regarded as due to cerebral hemorrhage or a thrombosis of the middle cerebral artery. The patient died five days later. Necropsy showed congestion of liver, spleen, and lungs, and typhoid ulceration of the ileum; apparently typhoid in the third week.

At necropsy a thrombus was found in the artery of the right Sylvian fissure and microscopically the areas supplied by this artery were found to have undergone softening with the presence of a great many fatty bodies or cells and cellular debris.

A STUDY OF THE BLOOD OF PATIENTS WITH PULMONARY TUBERCULOSIS UNDERGOING SANATORIUM AND TUBERCULIN TREATMENT.

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and

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The following study of the blood was made at the Adirondack Cottage Sanitarium between the years 1904 and 1911. Some of the work was for a time included in the ordinary routine work of the institution and other parts of it were taken up as separate investigations. Dr. Lupton, who was engaged upon the work at first, to our great regret, died before it was completed. The observations have been made as carefully as possible, and our work has been influenced by the excellent monograph of Kjer-Peterson, to which reference is made later.

ERYTHROCYTES.

Number per Cubic Millimeter. The number of red blood cells per cubic millimeter appears from a study of 273 cases to be slightly above normal. The figures for these cases were:

	Incipient.				Moderately Advanced.			
	Cases.	Average.	Median.	Mode.	Cases.	Average.	Median.	Mode.
Male .	77	5,630,000	5,500,000	5,000,000	63	5,710,000	5,500,000	5,000,000
Female	68	5,277,000	5,000,000	4,500,000	65	5,316,000	5,000,000	5,000,000

These cases when charted in the form of a frequency curve show for the incipient cases that the mode[1] for women is 4,500,000, for men slightly over 5,000,000. For the moderately advanced cases the mode for both men and women appears to be 5,000,000. The curves in both instances show a rather sharp rise and a slightly less rapid fall. The median and the mode differ slightly, the former being slightly higher than the latter.

Relation to Pulse Rate. The patients were divided into groups, accordingly as the erythrocytes ranged from 4,000,000 to 4,499,000

[1] The mean is the average value (or "average"); the median the middle-most value when the values are ranged in order of magnitude; the mode is the value which occurs most frequently. When the median and the mode coincide the mean gives an exact idea of the frequency relation; when they are widely separated the mean is of far less and often of uncertain value.

and so on up to 7,500,000, and in each group was placed the number of patients with a pulse rate of 80 to 89, 90 to 99, and so on to 120. The maximum for seven days on admission gave the average. The following conclusion was reached, namely, that the number of erythrocytes in incipient tuberculosis in males is smaller than in moderately advanced tuberculosis for pulse rates below 100. In women the same holds for any pulse rate. There were 128 men and 118 women studied.

Relation to Color Index and Color of Mucous Membranes. In calculating the color index 5,000,000 is used as a standard in both incipient and moderately advanced cases. If for women 4,500,000 is used as the standard the color index will be 10 per cent. higher. The mucous membranes were classified as follows: good color, slightly anemic, anemic, and slightly cyanotic, according to the appearance at examination on arrival. No relation was found to exist in 237 cases.

Relation to Hemoglobin and Color of Mucous Membranes. The cases were divided according to the number of erythrocytes into groups of 1,000,000 from 4,000,000 up to 7,999,000. They were arranged in these groups according to the percentage of hemoglobin, 60 to 69 per cent., 70 to 79 per cent., and so on. The color of the mucous membranes was classified as above. Nothing was learned in a study of 130 incipient and 107 moderately advanced cases.

Relation to Extent of Physical Signs. The following cases were studied:

	Turban.	I.	II.	III.
Males		85	28	5
Females		81	32	6

The results were tabulated, but showed little variation between the two groups.

HEMOGLOBIN.

The hemoglobin was measured on admission by the Talquist scale and occasionally with the von Fleischl instrument in 272 patients with the following results:

	Incipient.		Moderately Advanced.	
	Cases.	Average per cent. Hb.	Cases	Average per cent. Hb.
Males	77	92	62	91
Females	68	89	65	88

The results in the incipient and moderately advanced stages for males and females as well as the totals in each group were charted. The mode in every case but one was 90 per cent., the moderately advanced male group having 95 per cent. as the mode.

LEUKOCYTES.

Number per Cubic Millimeter. In all 275 cases were studied, with the following results:

	Incipient.				Moderately Advanced.			
	Cases.	Average.	Mean.	Mode.	Cases.	Average.	Mean.	Mode.
Male . . .	68	10,074	9000	8500	63	10,443	10,000	10,000
Female . . .	69	9,425	8000	7000	65	10,642	10,000	8,000

When a frequency curve for these cases is charted it is seen that for incipients the mode is between 7000 and 8000, with a sharp rise from 4000 and a more gradual decline to 16,000. The males and females each exhibit a less marked mode at 8000 to 9000 and 7000 respectively. For the moderately advanced cases the mode lies between 8000 to 10,000, with a sharp rise from 5000 and a gradual decline to 20,000. The males and females each exhibit a fairly well-marked mode at 10,000 and 8000 respectively. These figures all relate to counts made on admission, as the counts made on discharge are too few for any inductions to be drawn, but would seem to indicate that the curves are much flatter.

Relation to Duration of Disease. The cases were tabulated according to the length of illness, 0 to 5 months, 6 to 11 months, and so on to 120 months. The leukocytes were grouped into thousands from 5000 up to 21,000, and an attempt made to see what effect duration of disease had upon the number of leukocytes. There seemed to be no relation in 107 cases.

Relation to Weight. Incipient and moderately advanced cases, male and female, were arranged in groups, accordingly as their weights were above or below standard, allowing ten pounds to each group. The cases in each group were arranged also according to the thousands of their leukocytes, 5000 to 21,000. Digestion was unimpaired and general condition good for all cases. Nothing was learned from this study of 168 cases.

Relation to Tubercle Bacilli in Sputum. The cases were arranged accordingly as tubercle bacilli were present (1) previous to admission, (2) at admission, (3) during residence, and grouped according to the number of their leukocytes, 5000 to 21,000. There was apparently no relation in 169 cases.

Relation to Condition on Admission and on Discharge. A single leukocyte count of each patient was taken on admission and on discharge. The condition of each case was charted accordingly as the disease was apparently cured, arrested, or failed, and also whether tubercle bacilli were present or absent. The temperature was normal in all cases. No information was gained from 169 cases.

Relation to Temperature. The cases were divided according to the number of leukocytes into six groups above and below 10,000, and the number of cases in each group classified as incipient or moderately advanced. The leukocytes taken at other times of the day in this study were grouped separately from the 8 A.M. counts. No relation was found to exist in 63 afebrile and 36 febrile cases. There were no cases with vomica.

Differential Count. In a study of 60 cases the percentage of the polymorphonuclears varied inversely with the percentage of the mononuclears, and was lowest in the incipient, higher in the moderately advanced, and highest in the far-advanced. The eosinophiles followed the same rule as the mononuclears.

	No. of cases.	Polymorphonuclears.	Mononuclears.	Lymphocytes.	Large mononuclears.	Transitional.	Eosinophiles.	Mast cells.
Incipient	16	64.9	33.0	28.6	2.0	2.4	2.0	0.1
Moderately advanced	32	69.3	29.6	24.6	2.8	2.2	1.0	0.1
Far advanced . .	13	81.4	18.0	14.1	1.3	2.6	.5	0.04

Tuberculin. In order to determine accurately the effect of tuberculin upon the number of leukocytes, we have calculated the mean error in the number of leukocytes in several instances. The method that we have followed is that adopted by Kjer-Peterson in his interesting work upon the numerical relation of the leukocytes in pulmonary tuberculosis. While Kjer-Peterson calculated the mean error from different specimens taken at the same time from the same man, we calculated the mean error from ten counts of leukocytes in the same pipette. We also calculated the mean errors on twenty different estimations of leukocytes, and found these to be in 5 cases, 3 per cent. of the number; in 11 cases, 4 per cent. of the number; in 2 cases, 5 per cent. of the number; in 2 cases, 5 per cent. of the number. The lowest mean errors in percentages were found in the largest number of leukocytes and the highest mean errors in percentages were found in the smallest number of leukocytes. The number of leukocytes on which the mean errors were calculated was between 3400 and 10,780. Using the formula of Kjer-Peterson we find that the mean error in the observation of a single count is $\mathrm{Me} = \sqrt{\frac{(O_1 - O)^2 + (O_2 - O)^2 + \cdots\cdots (O_{10} - O)^2}{9}}$. The mean error of the mean of the ten observations or counts is $\sqrt{\frac{\mathrm{Me}^2}{10}} = \frac{\mathrm{Me}}{\sqrt{10}}$.

The calculation was made as follows:

Observation count O_n	Deviation from mean $O_n - O$	Squares of deviation $(O_n - O)^2$
34	−1	1
33	−2	4
36	+1	1
25	−1	100
36	+1	1
42	+7	49
34	−1	1
37	+2	4
37	+2	4
36	+1	1
Total 350		166

Mean (O) = 35.
Me = mean error.

$$Me^2 = \frac{166}{9}.$$

$$Me = \sqrt{\frac{166}{9}} = 4.298.$$

Now 4.298 is 12 per cent. of 35, consequently the mean error in these observations was 12 per cent., which is much higher than Kjer-Peterson's (8 per cent.).

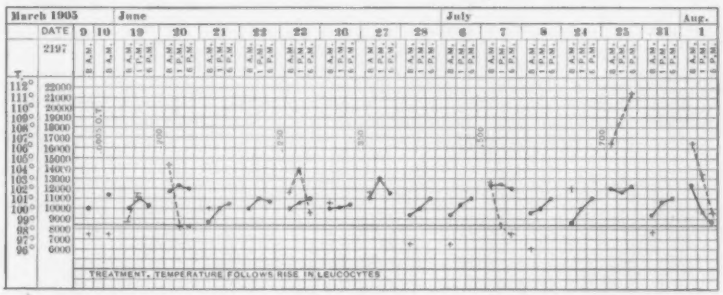


CHART 1.—Tuberculin treatment with O. T. Female, aged twenty-one years. Essential increase of leukocytes only after 700 mg. Lines with dots represent temperature; lines with crosses, leukocyte counts.

As Kjer-Peterson's studies of the number of leukocytes in the blood of women would seem to indicate that the number of leukocytes is not constant from day to day at any time of the day, varying from 3000 to 12,000, our observations have been confined to leukocytes in the blood of men, for the number of leukocytes in the blood of healthy men was found to be constant early in the morning, immediately upon awakening. The morning specimens were taken after bathing and dressing, at 8 A.M., before breakfast.

In 14 instances where specimens were taken first in bed and again after bathing and dressing in the morning, the difference between the two counts was not larger than the unavoidable error in observation in 12 instances. In 2 cases where the number of leukocytes was smaller after the bath the differences are respectively 18 per cent. and 17 per cent. However, the differences on the whole may not be larger than the differences caused by errors in observations and an unavoidable small inconsistency in the number of leukocytes in the blood taken in bed on different days, early in the morning. It would seem to appear, therefore, that it did not make any considerable difference in the number of leukocytes whether the counts were taken before or after bathing and dressing in the morning.

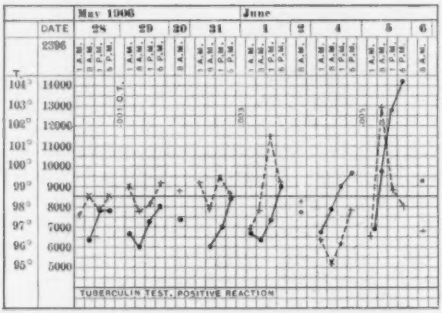


CHART 2.—Positive tuberculin test. Male, aged thirty years. Symptoms suggesting pulmonary tuberculosis. Essential increase of leukocytes after 5 mg. Lines with dots represent temperature; lines with crosses, leukocytes.

Tuberculin Treatment. The forms of tuberculin employed in the study were Koch's Old Tuberculin and the Bacillary Emulsion in varying doses up to 1000 mgs. of O. T. and 5 mgs. of B. E. The specimens of blood were taken at 8 A.M. after bathing and dressing, 1 P.M. and 6 P.M. before meals, and continued for many days during the course of treatment. In other cases, specimens were taken for five or six days in each month, at the same hours, and continued two to four months. We came to the conclusion from this study that there is no difference between O. T. and B. E. in their effects upon the leukocytes; that all doses may markedly increase the number of leukocytes; that there is little or no suggestion of leukopenia unless it comes shortly after the injection of tuberculin; that for a considerable increase in the dose there appears a considerable increase in the number of leukocytes corresponding to the increase in temperature, and that there may occur a rise in temperature without an increase in the number of leukocytes, and *vice versa* an increase in the number of leukocytes without a

rise of temperature. These studies were made after doses of tuberculin given at 8 to 9 P.M. When tuberculin is given at other times of the day it has not seemed possible to draw the same conclusions from our studies of the leukocytes. These observations were based on a study of 22 cases.

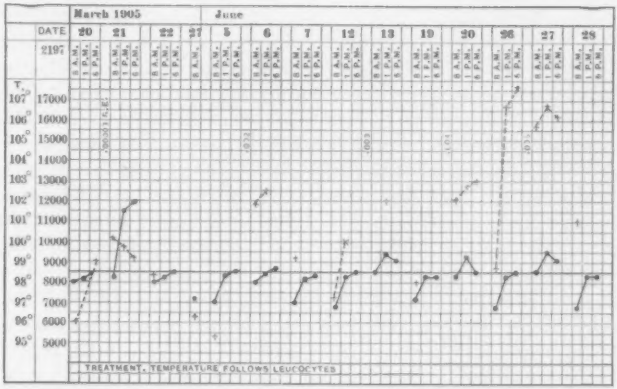


CHART 3.—Tuberculin treatment with B. E. Male, aged twenty-two years. Essential increase of leukocytes after last two doses of tuberculin. Lines with dots represent temperature; lines with crosses, leukocyte counts.

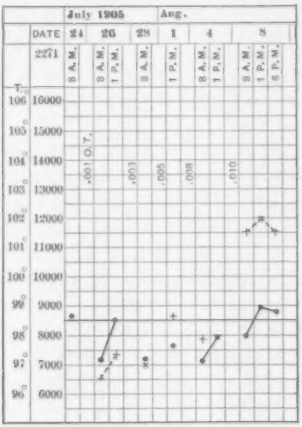


CHART 4.—Negative tuberculin test. Male, aged thirty-one years. Pulmonary abscess. Essential increase of leukocytes following 10 mg. of tuberculin. Lines with dots and crosses have the same significance as in preceding charts.

The Tuberculin Test. The tuberculin used was Koch's Old Tuberculin in doses of 0.5 to 10 mgs., given subcutaneously at

8 P.M., and specimens were taken at 1 A.M., 8 A.M. and 6 P.M. during the course of the test. The temperature was also taken at these hours. In order to determine whether there was a leukopenia following the dose and whether the temperature preceded or followed the rise in leukocytes when the patient reacted, tuberculin was given to another series of test cases at 8.30 A.M. and specimens were taken daily at 8, 10.30, 11.30 A.M., and 12.30, 1.30, 6 P.M. until reaction occurred. Specimens of blood were taken at the same hours on the day preceding the injection. It appears in this study that a reaction to tuberculin is almost always accompanied by a temperature. This followed in all these cases. During the period of reaction there is always an essential increase in the number of leukocytes when accompanied by a considerable increase in temperature; that the increase in leukocytes and temperature is usually closely connected; that the increase in leukocytes begin four to five hours after injection, and is closely followed by the rise in temperature. There was no evidence of a leukopenia observed in any of these cases. The rise of leukocytes is generally followed by a decrease in the number of leukocytes during the day after the first dose which causes an increase. In case of a repetition of the same dose or a slight increase of the dose the increase of the number of leukocytes corresponding to a considerable increase in the temperature appeared to be smaller and extended over a longer time. The counts taken at 1 A.M., four to five hours after injection, showed neither a leukocytosis nor a leukopenia. There were 27 cases studied.

NEUTROPHILIC BLOOD PICTURE OF ARNETH.

In 1904 Dr. Joseph Arneth endeavored to draw certain diagnostic and prognostic conclusions from hematological findings in pulmonary tuberculosis. His method depends upon an analysis of the nuclei of the neutrophile leukocytes, which constitute the bulk of the human white blood cells. He employed the usual dry smear with Ehrlich's triacid stain and divided the leukocytes into five classes according to the number of nuclei to a cell. His classification is arbitrary and somewhat complicated, and for a further description the reader is referred to his monograph.[2]

His original method was followed in a few cases, but it proved too laborious, and the subdivision of each class, accordingly as the nucleus was round, moderately indented, or deeply indented, was discarded, and we simply made divisions into five classes according to the number of nuclei to the cell.

Hastings' and Wright's stains were used and 200 cells counted.

[2] Die Neutrophilen weissen Blutkorperchen bei Infectionskrankheiten, Jena, 1904.

Date	Hour	1	2	3	4	5
8-1-08	8-AM	20	42	30	8	0
8-2-08	"	18	51	24	7	0
8-3-08	"	18	42	33	6	1
8-4-08	"	31	44	20	5	0
8-5-08	"	37	45	14	4	0
8-6-08	"	22	45	27	6	0
8-7-08	"	20	47	33	0	0
8-8-08	"	38	43	12	7	0
8-9-08	"	24	53	19	4	0
8-10-08	"	17	44	31	8	0
8-11-08	"	21	52	24	3	0
8-12-08	"	19	49	28	4	0
8-13-08	"	14	53	26	7	0
8-14-08	"	34	42	22	2	0
8-15-08	"	11	46	34	7	2
8-16-08	"	9	47	39	5	0
8-17-08	"	22	48	26	3	0
8-18-08	"	24	55	18	3	0
8-19-08	"	12	52	31	5	0
8-20-08	"	13	48	33	6	0
8-21-08	"	12	60	23	3	0
8-22-08	"	7	55	30	7	1
8-23-08	"	8	51	31	9	1
8-24-08	"	19	46	32	2	1
8-25-08	"	18	45	34	3	0
8-26-08	"	26	56	18	0	0
8-27-08	"	14	52	30	3	1
8-28-08	"	22	47	22	8	1
8-29-08	"	15	47	33	5	0
8-30-08	"	17	50	24	2	1
8-31-08	"	18	54	26	2	0
10-8-08	"	21	52	15	11	1

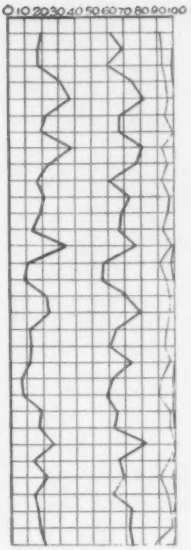
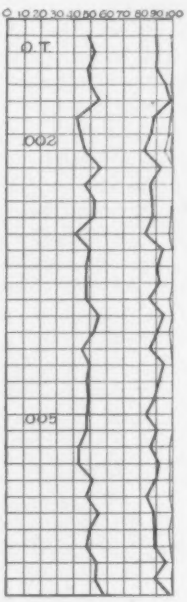


Table showing variation of the Arneth counts in an incipient case of tuberculosis.

Date	Hour	Leucocyte	Arneths Count 1	2	3	4	5
2-8-08	8-AM	6800	49	41	9	1	0
"	10-30 "	6300	53	38	9	0	0
"	11-30 "	6000	49	41	10	0	0
"	12-30PM	7800	50	46	3	1	0
"	1-30 "	6400	55	44	1	0	0
"	6- "	8400	42	47	10	1	0
2-9-08	8-AM	6800	44	43	11	2	0
"	10-30 "	6533	47	36	12	4	1
"	11-30 "	9000	56	37	7	0	0
"	12-30PM	8900	47	40	13	0	0
"	1-30 "	9200	53	35	12	0	0
"	6 - "	10300	52	36	11	1	0
2-10-08	8-AM	8200	41	42	15	2	0
"	10-30 "	7200	50	44	6	0	0
"	11-30 "	9133	48	43	9	0	0
"	12-30PM	8266	48	44	8	0	0
"	6 - "	8133	48	38	12	2	0
2-13-08	8-AM	8600	56	39	5	0	0
"	10-30 "	7500	52	39	7	2	0
"	11-30 "	7333	45	41	13	1	0
"	12-30PM	6600	50	45	5	0	0
"	1-30 "	4000	48	44	8	0	0
"	6 - "	5333	49	41	10	0	0
2-14-08	8-AM	6333	48	36	16	0	0
"	10-30 "	6133	48	43	9	0	0
"	11-30 "	5425	43	44	11	2	0
"	12-30PM	6400	43	49	6	2	0
"	1-30 "	10900	52	38	8	2	0
"	6 - "	10750	48	36	14	2	0
2-15-08	8-AM	7800	55	34	11	0	0
"	10-30 "	8000	50	39	9	2	0
"	11-30 "	7266	48	41	9	2	0
"	12-30PM	6400	54	42	4	0	0
"	1-30 "	5550	53	36	10	1	0
"	6 - "	6400	58	39	3	0	0



Abd. Admond R.

Table showing effects of tuberculin on the total leukocyte and Arneth count of a moderately advanced case of tuberculosis.

In the tuberculin treated cases smears were made at 8 A.M. daily for one week, and in 2 cases daily for one month. In the tuberculin test cases, smears were taken six times daily, at 8, 10.30, 11.30 A.M., 12.30, 1.30, 6 P.M., until the patient reacted. From a study of 50 cases the following conclusions appear probable:

1. The nuclear picture is of no practical value in determining the dose of tuberculin for therapeutic purposes. In 2 cases which were followed daily for one month the blood curve showed no definite relation to the dose, and at no time made any appreciable deviation to the left or to the right.

2. In the course of the subcutaneous tuberculin test the blood picture is apparently not influenced until the stage of reaction occurs, when there may be a slight shifting to the left. This was not constant in all our cases.

3. In tuberculosis with extension of the infectious process there is a marked and permanent deviation of the blood picture to the left, indicating a grave prognosis.

BLOOD PRESSURE.

The instruments used were the Erlanger and Janeway sphygmomanometers, with a cuff 12 to 15 cm. wide. The blood pressure of all patients was taken a day or two after their arrival at the Sanitarium. A great number, however, had been in the mountains some weeks before being admitted. All patients were at rest, flat on their backs, on a couch, for at least five to fifteen minutes before the observation was made. Three to five records were taken, and if the variation did not exceed 4 mm. of mercury the average was entered as the blood pressure. The diastolic records are not as numerous as the systolic, owing to some imperfection developing in the Erlanger machine before our work was completed.[3] The blood pressure of many of the same patients was taken again at discharge, which occurred some six months after their admission. At an altitude of 1600 feet we found the blood pressure to be:

Incipient.

	Cases.	Average.	Median.	Mode.
Male	63	127 mm.	128 mm.	122 and 133 mm.
Female	75	121 mm.	118 mm.	112 and 128 mm.

As there are two modes the mean or average does not give us in this series of cases the true systolic level and consequently the systolic pressure lies between 120 to 125 and 130 to 135 mm. for males and between 110 to 115 and 125 to 130 mm. for females.

[3] Owing to an imperfection in the Erlanger instrument the diastolic pressure was taken only in 84 patients. In 32 incipient cases it was found to be: males 86 mm., females 82 mm. In the moderately advanced cases: males, 85 mm., females 82 mm.

Moderately advanced.

	Cases.	Average.	Median.	Mode.
Male	118	126 mm.	125 mm.	125 mm.
Female	73	121 mm.	120 mm.	120 mm.

In this series of cases as the median and mode coincide the mean or average value gives us an exact idea of the true systolic pressure.

In the tuberculin treated cases (25) no appreciable effect was noted even after the largest doses (800 to 1000 mg. of tuberculin) had been given. The blood pressure of these cases was taken morning and evening and continued twice daily for two or three weeks. There were 329 cases studied.

TREATMENT OF ACNE WITH STOCK AND AUTOGENOUS ACNE BACILLUS VACCINE.

By E. D. Lovejoy, A.B., M.D.,

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Vaccine treatment in pustular dermatoses has now been in fairly extensive use for some time and varying results have been reported. By far the greater number, however, have been cases treated by vaccine of the two varieties of the staphylococcus, namely the albus and aureus.

It is now practically a recognized fact that cases of furunculosis and sycosis are cured much more rapidly by vaccine treatment than by any of the older procedures. Especially is this true in those resistant, long-standing cases of furunculosis, in which the disease is readily checked by a few injections of a stock vaccine, and a lasting immunity is produced by a continuation of the treatment.

In sycosis barbæ vaccine is of most benefit in clearing up those deep, indurated, nodular masses in which the infection has extended down into the subcutaneous tissues and the course of the treatment is much shortened by combining injections with the usual lotions and ointments.

The results obtained by the use of staphylococcus vaccine in acne vulgaris were not as successful as in the other pustular conditions. In the chronic cases some improvement resulted but the disease soon recurred, while in the acute cases where the outbreak consisted only of comedones and small superficial pustules, the results were practically negative. The present method of using a combination of an acne and staphylococcus vaccine has proved

much more successful in that it combats both of the infecting agents at once.

From results obtained, as well as from reports by Fleming, Gilchrist, Engman and others, there is no doubt that the acne bacillus plays a large part in the infective processes of acne, but that it is the primary cause of the disease and produces the hyperkeratosis of the outlets of the ducts and the hypertrophy and loss of function of the glands themselves is not accepted by all observers. Though the fact that the bacillus can be found in smears from all cases of acne and plays an equal, if not a greater, part than the staphylococcus is well recognized. For like the staphylococcus the acne bacillus can be found in the skin of individuals who are not, nor ever have been sufferers from the disease, in fact, it can be demonstrated in almost pure culture from the plugs expressed from the follicles along the inner border of the ala of the nose whenever an oily seborrheic condition is present.

The acne vaccine used in the following series of cases was prepared in the Cornell Laboratory of Clinical Pathology after the method published in the *Journal of Cutaneous Diseases*, February, 1911. The organism is grown on 2 per cent. glucose agar slants under anaërobic conditions, transplants being taken from the original growths whenever pure cultures were not obtained.

As regards technique it will be found much simpler not to combine the emulsions, but to use them from separate bottles, as in this manner the strength of the dosage can be easily regulated, the desired amount of the acne vaccine being drawn up into the barrel of the syringe and then the staphylococcus vaccine added to it and the whole quantity given in one injection.

The following reports are taken from some 50 cases of acne, most of them being well advanced and showing indurated and pustular lesions. Some of them are from private practice, the rest from Dr. Elliot's clinic in the Cornell Dispensary.

About one-third were treated by staphylococcic vaccine alone, of the remainder some were treated by autogenous vaccine, while for the rest a polyvalent stock emulsion was used made up from cultures obtained from the first series. In all cases smears were taken from the lesions and stained with Gram's stain to determine the presence or absence of the acne bacillus. In every case they were present, although in varying numbers. The constitutional treatment was simple and consisted of the correction of any digestive disturbance and regulation of the bowels, while locally a mild antiseptic lotion was employed to prevent a surface spreading of the infection.

The injections were begun with a dosage of 3,000,000 acne bacilli and 150,000,000 staphylococci, the same amounts being practically continued throughout the course of treatment, in no case a larger amount than 5,000,000 acne and 250,000,000 staphylo-

cocci being reached. The frequency averaged about one injection every five days. Abscesses and the larger pustules were wiped over with alcohol and incised before treatment was begun and the comedones were removed as thoroughly as possible, the processes being repeated if they recurred.

The autogenous and stock vaccine cases were treated as nearly alike as possible in order to make a comparison of the results obtained and the cases in both series were selected from the more severe, long-standing infections.

Between the results obtained in the two series little remained to choose, in fact the most brilliant success of all was a young woman suffering from a very widespread, pustular acne of four years' duration, who had received the polyvalent stock vaccine. All the patients showed marked improvement but some of them were lost trace of toward the end of the treatment, and have not been seen since. One patient who had been under autogenous vaccine stopped treatment when the condition showed only improvement and obtained employment somewhere in the south where he was able to be out of doors a good deal, but the improvement in general health completed the cure begun by the vaccine and he has remained free from lesions ever since. The immunity in the case of the young woman does not seem to have been so lasting, for her work was in a factory and the hygienic surroundings were not so good. At the time the course of treatment was stopped her face was entirely free from anything resembling an acne lesion. She remained away three months and then returned, four or five small superficial pustules having appeared on her forehead, one injection of the same vaccine was given, and she reported that the lesions promptly healed; the recurrence was, however, repeated in about two months, when she returned with a worse outbreak, but complaining of some menstrual disturbance which was corrected and a couple of injections of vaccine given. After that she remained in good condition for five or six months when a very slight outbreak occurred yielding easily to the vaccine. Since then she has apparently remained free from all lesions.

Another patient who was on stock vaccine, has shown himself most resistant to treatment. The case was one of long standing, and when first seen the condition had gone on to an extensive abscess formation on one side of the face. These abscesses were incised at the beginning of treatment, and at first good progress was made and a partial immunity established, but it was easily broken down and the patient is still under treatment. Although there is considerable improvement over the original condition the result is far from satisfactory. The failure is undoubtedly due to the poor condition of the general health, for antedating the acne invasion there was a tuberculous joint infection and wasting of the muscles of one leg which probably keeps his power of resistance below par.

In considering the use of the acne vaccine treatment several facts appear evident. First, that the results obtained with the polyvalent acne stock were fully as satisfactory as those obtained with an autogenous vaccine, also that much more depended on a careful oversight of the case, from the clinical standpoint, regulation of the amount and frequency of dosage and general hygienic care of patients, than on the fact that the vaccines were autogenous. This, of course, is of great advantage in the employment of the injection treatment, for the acne bacillus is at best a slow grower and requires considerable time and care.

Another fact observed is that old emulsions produce a more rapid immunity with less danger of local reaction or anaphylaxis than those freshly prepared. Any emulsion carefully prepared keeps indefinitely if care is taken to prevent contamination. This fact has also been observed in vaccine injections against typhoid, in which it has been shown that an old emulsion produces slight if any, constitutional reaction, as compared with fresh cultures.

A FATAL CASE OF TETANY WITH AUTOPSY FINDINGS SHOWING HEMORRHAGES IN THE PARATHYROID GLANDS.

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It is an acknowledged fact that tetany occurring after an operation for the removal of goitre is to be attributed to the removal of the parathyroid glands along with the thyroid gland, and that tetany of children is due to disease of the parathyroid glands.

Whether non-operative or spontaneous tetany occurring in adults is due to disease of the parathyroid glands or not is a question which is not yet settled. Indeed, as yet we have only insufficient data for deciding this question, as only few cases of fatal tetany in adults with postmortem findings have been recorded.

It has been our good fortune to observe a case of tetany in an adult in which the clinical course and postmortem findings seem to leave no doubt as to the diagnosis.

The parathyroid glands, which were discovered in 1880 by Sandstroem, were forgotten for ten years and again brought to our attention by the French physiologist Glay in 1891. The vital importance of the parathyroid glands is very generally recognized.

These glands are found on each side of the thyroid gland and sometimes embedded in the same. The number and position of these little glands show great individual variation. They are more numerous in children than in adults, and are bean-shaped, flat, and slightly translucent, of a yellow-brown color, and never lobulated. Oftentimes it is hard to distinguish them from fat tissue. Microscopically they consist of epithelial cells. Hence the name "epithelial bodies" given them by Kohn.[1]

The cell masses are arranged in lobules or cell chains, between which a connective-tissue stroma runs which is less developed in younger individuals than in older ones. The bloodvessels are embedded in this stroma. The parenchyma consists of two different kinds of cells: The first are large polygonal and sharply outlined cells (cell type I), the protoplasm of which is colorless or slightly stained with eosin. Between these cells we find a deeper stained intercellular substance. The second type of cells are rich in chromatin (cell type II). They are indistinct in outline, and their scanty protoplasm is stained more or less with eosin. These cells are in some places closely packed together, giving the impression of being free nuclei. Koenigstein[2] has demonstrated on serial section that both types of cells are identical, and are different forms of the same cell, the differentiation having been produced by functional activity.

Petersen[3] demonstrated with methyl-green pyronin stain that cell type I is a cell resting, while cell type II represents the cell in functional activity. The epithelial cells in young individuals contain much glycogen, and later fat drops. A few colloid drops may be found.

The colloid production found in the thyroid gland is not found in the parathyroid glands. In older individuals the glands are infiltrated with many large interstitial fat cells, which develop from the perivascular connective tissue.

The physiological function of the parathyroid glands became known to us only in the last few years. After overcoming many difficulties, Glay, who made the first experiments on rabbits, showed that the animals died of tetany after total extirpation of the thyroid glands with all the parathyroid glands. On extirpation of the thyroid gland without the parathyroid glands death did not occur. Glay believed that the parathyroid glands can perform the function of the thyroid gland, but on account of the irregular results from his experiments he could not give a clear idea of the function of the parathyroid glands.

Mousson took up Glay's experiments and criticised the same. He experimented on dogs, and reached the conclusion that the extirpation of the thyroid gland without the parathyroid glands

[1] (Collective references.) Ergebn. d. Anat. Entwicklungsgesch., 1899, ix.
[2] Wien. klin. Woch., 1906.
[3] Virchow's Arch., clxxiv, 1903.

does not cause tetany, but causes a chronic cachectic condition which the animal outlives. The extirpation of the parathyroid glands alone cause tetany, with fatal result.

This strict dualism of Mousson has been confirmed by a number of investigators: Vassale,[4] Genrali, Walbum, Pineless,[5] Erdheim,[6] Hagenbach, Iselin, and others. Therefore, two different diseases may be distinguished: tetany parathyreopriva and cachexia thyreopriva. The term tetany thyreopriva is misleading, and is only a collective name for undifferentiated diseases. Without going into details of further experiments on animals, we may compare these with our clinical experience with human beings. We know that after thyroidectomy on human beings, tetany is observed, and it is believed that this is due to the removal of the parathyroid glands. The experimental experience on animals has demonstrated the same.

Erdheim was the first to throw light on the subject of operative tetany after thyroidectomy. He examined in serial sections the throat organs of two individuals dying after thyroidectomy, and could not find any trace of the parathyroid glands. The remaining portion of the thyroid was sufficient, it seemed, to protect the individual from cachexia thyreopriva. The result of Erdheim's investigation is in accord with animal experiments. Since surgeons have learned to avoid removal of all the parathyroid glands, tetany is very rare after thyroidectomy. Absolute proof as to the different functions of the parathyroid glands and the thyroid gland is seen in cases of so-called athyroidismus, or complete congenital defect of the thyroid. Here we have the typical picture of cretinism. In the cases which came to autopsy the parathyroid glands were found, but there was no trace of the thyroid gland. Experimental experience and the clinical observations are in harmony, and all argument against the functional activity of the parathyroid glands can now be disregarded.

We now approach the important and, to us, the most interesting question of the relationship of idiopathic, infantile, and adult tetany.

Erdheim was the first to discover hemorrhages in the parathyroid glands in cases of infantile tetany.

Yanase[7] was the first to study this subject extensively, which he did from the cases in Escherich's clinic. He examined the parathyroid glands of 89 children coming to autopsy without any

[4] Rivista experim. di freniatr., 1897; Arch. ital. de Biol., 1898; Münch. med. Woch., 1906, Nr. 33.

[5] Sitzungsber d. Kais. Akad. d. Wiss. in Wien, 1903, Abt. iv; Mitt. a. d. Grenzgeb. d. Med. u. Chir., 1904; Arb. a. d. Wien, Neurol. Inst., Nr. 16; Arch. f. klin. Med., lxxxv, 1906.

[6] Wien. klin, Woch., 1901, Nr. 41; Zeit. f. Heilk., Abt. f. path. Anat., 1904; Mitt. a. d. Grenzgeb., 1906, xvi; Ziegler's Beitr., 1903, xxxiii; Sitzungsb. d. Wien, Akad. d. Wiss., 1907, Abt. iii (mathemat.-naturwissensch.).

[7] Jahrb. f. Kinderheilk., lxvii, 1908.

selection, ranging from birth to the fifteenth year. He found hemorrhages or remains of them in 33 children. In all, he examined 104 parathyroid glands and found 71 with hemorrhages. Therefore, hemorrhages in parathyroid glands are of frequent occurrence.

Yanase made systematic investigations of the electrical irritability in sick children, and at death he examined the parathyroid glands in 50 cases which came to autopsy; 30 of these showed a normal electrical irritability. At autopsy of these 30 cases the parathyroid glands were found normal, and no hemorrhages were found. In contrast, he found increased anodal electrical irritability in 22 children; and in 12 (55 per cent.) of these, hemorrhages were found in the parathyroid glands. Of 9 children under one year of age hemorrhages were found in each case. In the other 13 all of whom were older than one year, only 3 showed remnants of hemorrhages. Yanase came to the conclusion that in the remaining 10, hemorrhages had occurred which could not be found at the time of autopsy.

The latest investigations of Haberfeld[8] has thrown some light on such cases of infantile tetany where no hemorrhages were found at autopsy. He found in these cases more or less marked atrophy, with or without remnants of hemorrhages, such as pigment or cyst formation. His thorough histological examination of these parathyroid glands has shown that hemorrhages no only destroy the histological structure, but also inhibit the development of the parathyroid glands and lead to a hypoplasia.

The reason that no symptoms of tetany were observed at the time of hemorrhage which generally occurs at time of birth is explained by Escherich,[9] that the central nervous system of the newborn is insensitive to tetany toxin. The susceptibility to tetany begins about the third month. This theory seems to be supported by Quest,[10] who found that the brain of the fetus contains the greatest amount of calcium, which decreases with the age of the child, and the irritibility of the brain is increased with the decrease of calcium.

The opinion of Escherich and Quest is in accordance with the recent investigations of Voetglin and McCallum,[11] who revealed the fact that the calcium excretion of parathyroidectomized animals was increased and the tetany attacks could be eliminated by calcium salts injection.

Therefore, we conclude that infantile tetany is due to insufficiency of the parathyroid glands produced by hemorrhages.

Before discussion of the adult tetany we wish to report our case, that of a man, aged twenty-six years, a Russian, speaking little English, a butcher by occupation. He was admitted to the Allegheny General Hospital, May 25, 1909, in the service of

[8] Virchow's Arch., cciii, Heft 2.
[9] Die Tetanie der Kinder.
[10] Jahrb. f. Kinderheilk., 1905.
[11] Jour. Exper. Med., 1909, xi, No. 1.

Dr. McNaugher, to whom we are under obligation for permission to examine the patient and to make this report. One week after admission, and two weeks after the onset of the disease, the patient died.

He had never been sick before the present illness except with an attack of influenza. His employer reports that he drank large quantities of pure alcohol and smoked cigarettes excessively. There was no history or sign of an infected wound.

Two weeks before admission the patient fell off a porch, a distance of five feet, and sustained an injury to the spine in the region of the third or fourth lumbar vertebræ. One week after this accident (two weeks before his death) he developed the first symptom of his fatal illness, an attack of rigidity in the arms and legs. These attacks came with increasing frequency and severity. When he was admitted to the hospital the attacks of rigidity affected the arms, legs, and trunk, including the neck. These attacks were intermittent and accompanied by considerable pain in the arms and legs, especially the latter. On admission, he complained much of pain in his legs. He asked to have his knees flexed, as he said this afforded great relief. It was noted that the patient became quite cyanotic during spasms, and that he perspired profusely.

May. 25. Sputum was negative for tubercle bacilli. The urine showed a slight trace of albumin and some hyaline casts.

May 28. Lumbar puncture was made, but resulted in a dry tap. The Wassermann test was negative.

Blood: Hemoglobin, 75 per cent.; red blood cells, 4,100,000; leukocytes, 7600. Differential count: Polynuclear neutrophiles, 78 per cent.; large lymphocytes, 11 per cent.; small lymphocytes, 10 per cent.; monocytes, 1 per cent.

On May 28 the patient was examined by Dr. Diller. The patient's mental condition seemed clear. Handling the arms and legs produced generalized spasms, which kept up for a minute or two. Pressure on the legs, even light pressure, caused a generalized spasm affecting the arms, legs, and trunk, and neck muscles. His position became one of opisthotonos; arms and legs were hyperextended. The legs were more involved than the arms. There were no spasms of the fingers and toes, and none of the face muscles; but pressure over the seventh nerve produced facial spasm. The masseters (especially examined) were free from spasms. Kneejerks were slightly increased; there were no Babinski signs.

The same day the eye-grounds were examined by Dr. A. C. Blair and found to be normal. The nurse noted that the spasms came particularly when the patient attempted to raise his knees; and in his examination, Dr. Diller noted the same. The spasms now came spontaneously without handling of the limbs, but they were especially provoked by handling or pressure anywhere on the legs or arms, especially the former.

On June 1 the patient was again examined by Dr. Diller. Prior to this his temperature had been normal, but his pulse had been accelerated, ranging from 90 to 120. On this day his temperature suddenly shot up to 103°, and he died a few hours later. For the preceding three days he had had no spasms in the daytime, but they were especially severe at night. At this examination no spasms were produced by pressure of the arms and legs, even when made over the vessels and nerves, but there was resistance in both arms and legs to passive movements. There were no spasms of the masseters, but considerable stiffness of the neck muscles.

The patient died a few hours after this examination was made.

The autopsy showed an external fibrous pachymeningitis, chronic leptomeningitis, edema, and hyperemia of the brain, hyperemia of the spinal cord, bilateral adhesive pleuritis, spleen slightly enlarged, hemorrhagic gastro-enteritis, hyperemia of kidney and liver, with slight parenchymatous cloudiness.

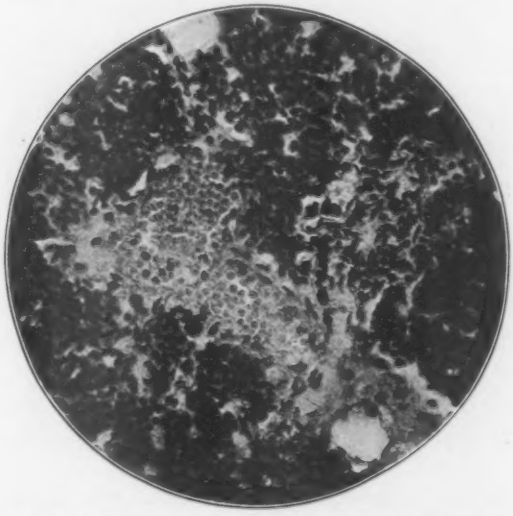

FIG. 1.—Ruptured bloodvessel with perivascular hemorrhage. Leitz, objective 5; ocular 4.

Special attention was given to the examination of the parathyroid glands, which in this case were found without any difficulty. Two were on the right side and one on the left side. The two former measured 10 mm. long by 3 mm. broad, and 9 mm. long by 2.5 mm. broad, while that on the left side measured 11 mm. by 3 mm. broad. Macroscopically the glands were opaque and brownish in color, with red punctations having the appearance of hemorrhages. The symptoms of tetany could not be accounted for by

the macroscopic changes found at autopsy, except for the suspicion of hemorrhages of the parathyroid glands. The pachymeningitis, leptomeningitis, and gastro-enteritis were probably of alcoholic origin.

Microscopic Examination of the Organs. The changes in the three parathyroid glands were almost uniform. With low power, extensive infiltration of the parenchyma with large fat cells was noted, causing partial atrophy. The connective-tissue stroma was greatly thickened and divided the parenchyma into various sized lobules. Bloodvessels and capillaries were greatly distended. With higher power the parenchyma consisted largely of small, indistinctly outlined epithelial cells which were rich in chromatin

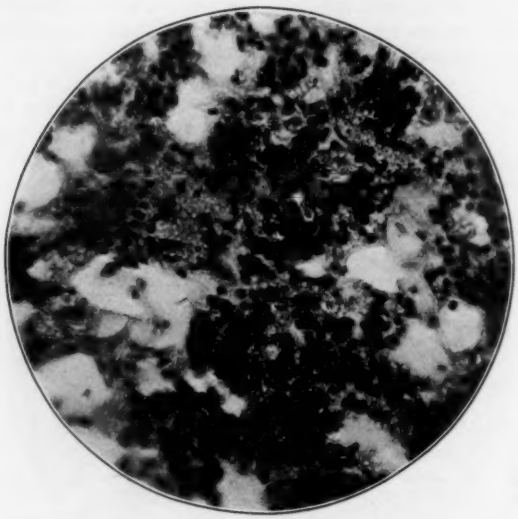

FIG. 2.—Parenchyma invaded by profuse hemorrhages. Leitz, objective 5; ocular 4.

(cell type II). The protoplasm stained bluish red. On the edge of the gland were found large, compact, distinctly outlined polygonal epithelial or oxyphile cells (cell type I). The protoplasm of these cells was finely granular, somewhat cloudy, and stained fairly well with eosin. The small intercellular mass stained more deeply with eosin. The nuclei of the cells were not so rich in chromatin as in cell type II, and as in some near the periphery. Some cells were without nuclei. Small groups of the same cells were found inside of cell type II.

The fine granulated appearance and the bluish-red color of cell type I and II suggested a slight parenchymatous degeneration. In

normal epithelial cells the protoplasm stains deep red with eosin, or is nearly colorless.

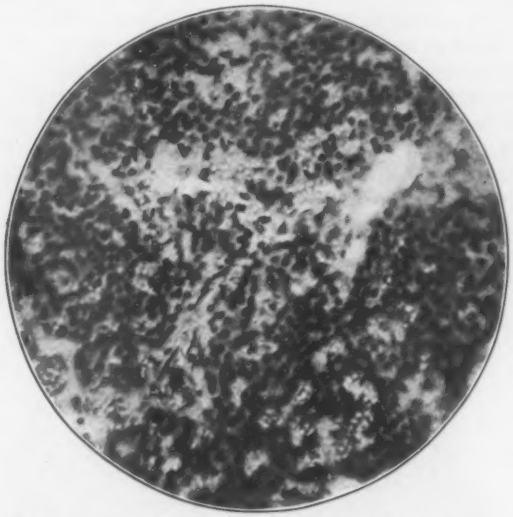

FIG. 3.—Same as Fig. 2. Leitz, objective 5; ocular 4.

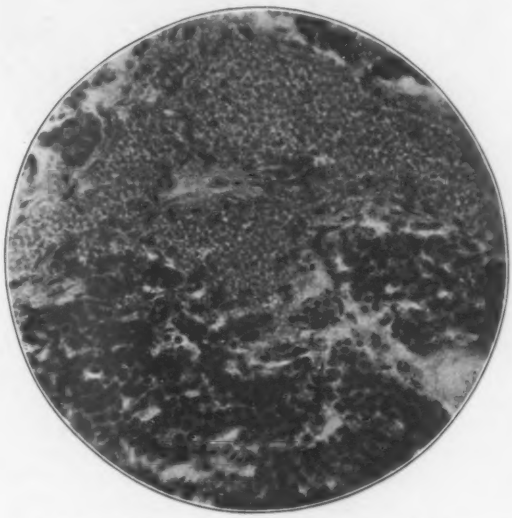

FIG. 4.—Same as Fig. 1. Leitz, objective 5; ocular 4.

The most interesting morbid change in the glands was the multiple hemorrhages which were diffused throughout one-half to two-thirds of the parenchyma in the form of small circumscribed areas. These hemorrhages could be traced to medium-sized blood-vessels. Larger perivascular hemorrhages, as found in the parathyroid glands of children, were not noted. The appearance of the hemorrhages was as follows: The red blood cells lay directly on the epithelial cells, which could be demonstrated with the Van Gieson stain. In contrast with the distended bloodvessels and capillaries fine, spindle-shaped endothelial cells were seen, deeply stained and at regular distances apart, which differentiated them from a hemorrhage. The fibrous adventitia of the vessels showed slight hyaline degeneration. Some vessels had very small lumina. The walls of the veins were much distended. In some places drops of colloid inside of glandular-like formations of epithelial cells were seen.

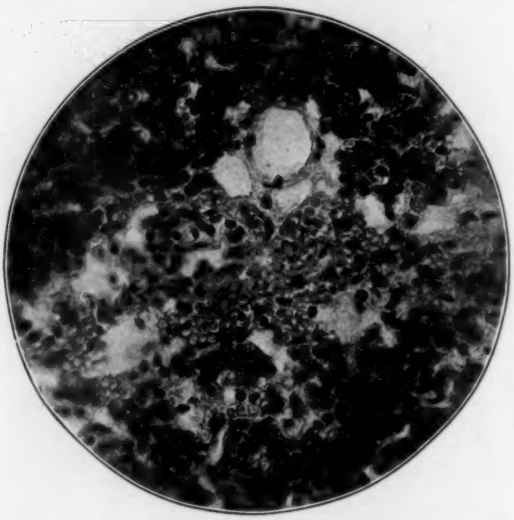

FIG. 5.—Same as Figs. 1 and 4. Leitz, objective 6; ocular 4.

Retrogressive changes of the hemorrhages, such as pigment, were not found, and this led to the conclusion that the hemorrhages must have been of recent origin.

The red blood cells presented a peculiar appearance. Some of them appeared deeply stained with eosin and were distinctly outlined. Others were merely visible and were stained very lightly. One received the impression that the hemoglobin had been extracted from them. We believe that this phenomenon is not due

to incomplete fixation. Petersen[12] has called our attention to this phenomenon. In his opinion the deeply stained bloodvessels are loaded with colloid, which is transported by the red cells through the organism. But we are not prepared to accept this view.

Microscopic Examination of the Central Nervous System. Numerous sections of the cortex and spinal cord, stained after Nissl, showed that the large pyramidal cells in the cortex were degenerated (absence of tigroid and vacuolation); some cells were without nuclei. The dentrites were greatly elongated. Practically the same change was found in the large cells of the anterior horns of the cord. No noteworthy change was seen in the white matter or the bloodvessels. The cells in the spinal ganglia were without change, and the peripheral nerves appeared normal.

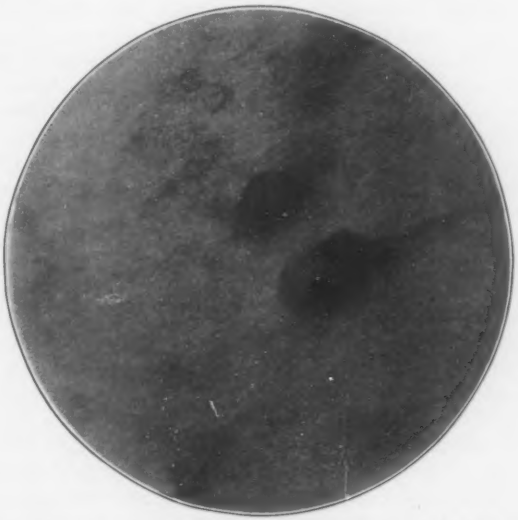

FIG. 6.—Large pyramidal cells of cortex of the frontal lobe of brain, stained after Nissl, showing prolongation of dentrite and absence of tigroid. Leitz, $\frac{1}{12}$ oil immersion; ocular 4.

The kidney and liver showed slight parenchymatous cloudiness, and their bloodvessels were greatly distended. The mucous membrane of intestine and stomach showed a few small hemorrhages.

In addition to our case, we review briefly the cases of tetany adultorum which can be divided into three groups.

Group I. Four cases of Haberfeld,[13] with typical clinical symptoms of tetany and changes of the parathyroid glands, found at autopsy. Case I showed amyloid degeneration of the small blood-

[12] Loc. cit.

[13] Loc. cit.

vessels and cyst formation of the large polygonal cells. Cases II and III showed hypoplasia, with cicatrix due to previous disease. Case IV, the first case of tetany gravidarum studied, showed hypoplasia and cicatrix of the parathytoid glands. This group may be named "hypothyroidismus."

Group II. One case of McCallum,[14] with typical clinical tetany, and no parathyroid glands found at autopsy (aparathyroidismus).

Group III. Three cases of Erdheim,[15] two of gastric tetany and one from brain tumor; one case of Koenigstein, carcinoma of the pancreas, all with typical clinical tetany, and no microscopic changes in the parathyroid glands.

The question arises as to whether any relation exists between tetany adultorum and the parathyroid glands. Cases under Group I and II are undoubtedly due to disease of the parathyroid glands. As above stated, the recent investigations of Haberfeld have proved atrophy of the parathyroid glands to be the chief cause of infantile tetany, and in all probability it is also the cause of tetany adultorum.

McCallum's case of aparathyroidismus stands isolated, and its explanation is difficult. The aparathyroidismus was either due to a congenital defect or a destructive lesion of the glands.

In our opinion it is not likely that it was due to a congenital defect, because the individual would not have lived so long without symptoms of tetany, but on account of their probable atrophic condition the parathyroids might have been overlooked. On the other hand, there might have been a lesion attended with complete destruction of the glands, thus causing tetany, or possibly there might have been an acquired serogen or histogen immunity to tetany toxin which has been demonstrated by animal experiments (Blum). Finally, the individual may have died from acute intoxication from tetany toxin, which could not be neutralized.

How may we account for the third group where no microscopic change of the parathyroid glands was found?

We believe that in such cases there was a hypoparathyroidismus due to atrophy of the gland without any other microscopic change.

The study of these cases was during the earliest investigations of the epithelial body, when the significance of the atrophy of the gland was not taken into consideration.

Our knowledge of the pathological anatomical changes in the parathyroid glands is limited. The latest investigations show that the parathyroid glands are very rarely diseased, at least we find in the literature very few observations of typical pathological changes.

Infiltration of the glands was found in leukemia, pernicious anemia, bacterial emboli with metastatic abscesses by Erdheim

[14] Die Beziehung der parathyreoiddrüsen zur Tetanie, Ztlbl. f. allg. Path. u. path. Anat., lxxvi, 1905.

[15] Loc. cit.

and Yanase; tubercular and syphilitic inflammation by Pepere,[16] Benjamin, Schmorl,[17] Koenigstein,[18] Carnot and Derion,[19] Verebely,[20] Stumme, Yanase; tumors, myoma, by Pepere; adenoma by De Lauti, Erdheim, Benjamin, and McCallum; lymphoma and angioma by Askanazy, Weichselbaum, and Verebely.

In all these cases these findings were accidental as no tetany was observed.

Hemorrhages in the parathyroid glands of adults without tetany were found by Erdheim in a man, aged forty-five years, who died of tuberculosis.

Pepere found hemorrhages in 2 cases: one, a girl, aged eighteen years, with a heart lesion and hemorrhages and cyst formation without pigment in one parathyroid gland; the other, a man, aged eighty-two years, without special clinical and anatomical findings, with fresh hemorrhages in one gland.

Verebely found rather recent hemorrhages in two glands, hematogen pigment in surrounding tissue, in a man, aged thirty-eight years, the cause of death is not mentioned.

Our case is the first in which an adult with tetany and hemorrhages of all parathyroid glands was observed. We believe the hemorrhages were all of recent occurrence, because no pigment was present. All three glands showed more or less primary atrophy of the parenchyma, followed by hemorrhages, which we believe were the indirect cause of the tetany.

Here the already atrophic glands were rendered insufficient by the hemorrhages.

In the infantile tetany we have first the hemorrhages which produce the atrophy of the glands.

According to Haberfeld[21] the diseased epithelial bodies are not alone responsible for the tetany, and a person could live for a long time with changed glands until disturbances, such as intestinal auto-intoxication, pregnancy, etc., would cause increased activity, which leads to tetany. Therefore, the insufficiency of the parathyroid glands is the chief cause of tetany.

He distinguishes between absolute and relative tetany. The absolute is derived from the absence of the epithelial body (operative tetany). The relative tetany is caused by increased metabolic products, which cannot be neutralized by the normal parathyroid glands or by a normal amount of metabolic products which cannot be neutralized by the insufficient parathyroid glands.

His latest investigations prove that changed parathyroid glands

[16] Leghiandole paratiroidee. Turin 1906; Arch. d. Anat. e di Embriol., viii, fasc. 2, 1909; Arch. de méd. expér., January, 1908.

[17] Münch. med. Woch., 1907.

[18] Ges. f. inn. Med. u. Kinderheilk., December 6, 1906.

[19] Compt. rend. dela Soc. de Biol., 1905.

[20] Virchow's Arch., 1907, clxxxvii.

[21] Die Epithelkörperchen bei Tetanie und bei einigen anderen Erkrankungen, Virchow's Arch., cciii, Heft 2.

plus increased metabolic activity is the chief factor in producing tetany, and "in infantile tetany this seems to be a law."

The disturbances are also the predisposing cause, the exciting cause being of unknown source.

Some investigators, especially Koenigstein, claim that the microscopic change in the gland is not sufficient to account for the clinical symptoms of tetany, but this opinion cannot be accepted.

All variations of the pathological process in the parathyroid gland lead to disturbance or decrease in their function.

A few referred diseases[22] of the parathyroid glands are not recognized by Erdheim and Haberfeld.

The hyperplasia of the gland found by Erdheim in osteomalacia remains to be studied. At present we can only say the diseased parathyroid glands cause tetany; as to other diseases, nothing has been demonstrated.

The real etiology of tetany is unknown, but it seems to be of an enterogenous origin. Without doubt the glands have the functions to avert and deintoxicate certain toxic products (autoneurotoxins), which are formed during metabolism, and which have a great affinity for the central nervous system. They also play the role of a protective apparatus for the central nervous system.

The clinical observation and pathological examination in conjunction with animal experiments have solved one of the most difficult problems of the ductless glands, and it is hoped that the real etiology of the tetania parathyreopriva will soon be made clear.

RHEUMATIC MYOSITIS.

By V. C. Rowland, M.D.,
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(From the medical Dispensary of the Lakeside Hospital, Cleveland.)

The particular phase of the large subject of muscular rheumatism considered in this paper is the relation of certain acute inflammatory conditions of the muscles to acute rheumatic fever. Chronic muscular rheumatism was fully described so long ago as 1816 by Balfour. Rheumatic involvement of muscles was also extensively studied by the Swedes, notably by Froriep, in 1843, and Helleday, in 1876. They, however, paid more attention to the chronic nodular condition and its treatment by massage. Considerable was written about the subject of acute myositis in the

[22] Eclampsia gravidarum (Vassale Zanfrognini), paralysis agitans (Lundborg), myasthenia gravis pseudoparalytica (Lundborg and Chvostek).

German literature in a period of about five years from 1892 to 1897. A number of series of acute cases, some epidemic in character were reported and the general consensus of opinion at that time was that they were infectious and identical with acute articular rheumatism or the same in an attenuated form. Some even claimed that many of the mild cases reported as dermatomyositis were of the same nature. There is little doubt that the condition has been variously diagnosticated, as the cases to be reported later well illustrate. The English literature on the subject is scanty. In the last year several articles have appeared, but like the Swedish writings, mostly with reference to the chronic muscular conditions under the name of fibrositis or nodular fibromyositis, which is described as independent of acute rheumatism or, indeed, any acute myositis. The French literature is also scanty. However, Rose,[1] under the heading of muscular cephalalgia, although describing the chronic fibrositic condition, refers to rare acute cases, which subside under salicylates and mentions acute muscular and articular rheumatism in the etiology.

In that period of interest (1892-1897) in the German literature, when these cases of acute myositis were more recognized than at any time since, the subsequent condition of a nodular infiltration (*Rheumatische Muskelschwiele*) was repeatedly described, sometimes in cases that had definite joint and muscle involvement at the same or different times, sometimes when only the muscle was involved. The infection of rheumatic fever may not be the only cause of the recently exploited fibrositis, especially if, as was was done by Luff, one includes under that heading such obviously different fibroid processes as Dupuytren's contracture.[2] But, the first question is whether a specific rheumatic myositis exists, that is, as a local manifestation of the generalized infection of rheumatic fever, the same as arthritis, phlebitis, or iritis. Various writers have inferred that the condition was an established fact (Kerr[3]), yet in the rather detailed classification of the inflammatory conditions of the muscles in Albutt and Rolleston's series, there is no mention of it. One case is described in Osler's series.

Before considering further the etiology of this condition the following cases from the Medical Dispensary of Lakeside Hospital may be reported:

CASE I.—This case is so typical that it may be reported at some length. Miss L. M. came to Lakeside Dispensary for the first time in 1897 complaining of blurred vision. A diagnosis of interstitial keratitis was made, and a few ridges on the teeth were regarded as confirmatory evidence of congenital syphilis. The clouding all cleared up after about four or five months of antisyphilitic

[1] Sem. Médicale, vol. xxxi, pp. 145, 156.
[2] Lancet, 1910, i, 712.
[3] Amer. Jour. Obst., vol. lxiii, p. 931.

treatment. One year later the same condition recurred in the other eye. This also cleared up with the same treatment. In the summer of 1900, the patient returned to the Medical Dispensary with a painful swelling of her left arm in the upper outer part. She had never had acute rheumatism or any infectious diseases that would have any bearing on the case, except the specific keratitis. Evidently the swelling was suspected to be an abscess, as it was incised in the dispensary. There was no suppuration and the wound healed rapidly, leaving the swelling the same. She was then given a course of antisyphilitic treatment without improvement. In August of 1900 she was sent into the hospital on the surgical service. A general anesthetic was given, and a second larger incision was made. The record of the operation reads as follows: "Over the periosteum was a layer of velvety gelatinous material. An attempt to curette this away failed, so all the tissue was chiselled away, down to firm bone. The wound was packed wide open. Subsequently it was noted that there was only rather free serous discharge from the wound, and that healing took place rapidly. The swelling was still distinct, although somewhat less at the time of her discharge, August 14, 1900.

After this experience she did not return to the hospital until the fall of 1911, when she appeared at the Medical Dispensary and related the following history: She went to a physician outside who treated her arm mostly by counter-irritation over a period of six months. The condition in her arm then practically disappeared, but left some indefinite soreness, for which she had several treatments by a masseur. All symptoms then disappeared and never again recurred in the same location. Several years later she had another swelling over the upper end of the sternum which was also incised to drain a supposed abscess, but no pus was obtained. In 1901, that is, previous to the second obscure swelling, she had a typical attack of acute articular rheumatism, and in 1904 a second, and in March, 1911, a third. With the last attack the right temporal muscle and the lower third of the quadriceps extensor femoris became swollen and painful. The patient thinks these swellings were of the same nature as those of the arm and sternum. The thigh swelling subsided promptly. The right temple, however, remained swollen, indurated, and painful, and caused constant headache. After enduring this for about two weeks, she appeared at the Medical Dispensary with the temporal muscle rounding out from the zygoma to the temporal ridge prominently enough to give a rather ludicrous contour to her otherwise small narrow face. At this time there was also marked chronic endocarditis with mitral insufficiency. She was given 15 grains of sodium salicylate four times a day, but returned in a few days unimproved. She was then given 15 grains every two hours with an alkali until toxic. The pain disappeared in one day. The swelling was nearly all gone in three

or four days, leaving some diffuse induration. Since then the swelling has recurred at intervals of three or four weeks, each time being relieved by salicylates. During October, 1911, the condition became more continuous and chronic, and reacted less favorably to salicylates. Since then she has been treated by massage, and is regarded as a case of indurative headache, as described by Edinger.[4]

A few other cases may be reported more briefly.

Case II.—Boy, aged fifteen years. Mother rheumatic. Patient had measles, mumps, pertussis, and diphtheria, but no tonsillitis or acute rheumatism until the present illness, which began with sudden pain in the wrist, shoulder, and ankle. After about two days the upper half of the right arm swelled up over night, and remained so for a week or more, during which time the joint symptoms had subsided. He was then admitted to the hospital with a temperature of 101°, the right arm the seat of marked swelling of a firm elastic character, skin redness, and so much pain and loss of power that the arm lay helpless at his side. Acute osteomyelitis was considered in the diagnosis. Two *x*-ray plates were negative. The blood examination was as follows: Leukocytes, 8,800; hemoglobin, 95 per cent.; Differential count: Small mononuclears, 21 per cent.; large mononuclears, 5 per cent.; polymorphonuclears, 69 per cent.; eosinophiles, 4 per cent.; transitionals, 1 per cent. He also had at this time a slight mitral insufficiency. He was rendered toxic with salicylates with prompt relief. The swelling subsided gradually. At the end of three weeks his arm was painless, but the biceps and the upper part of the brachialis anticus were rather thickened and indurated.

Case III.—Mrs. M. C., aged thirty-nine years. Two weeks after the onset of acute articular rheumatism involving both knees and ankles there appeared swelling and tenderness of the upper part of both thighs, more marked on the right, also tenderness in the lower two inches of the biceps muscle and its tendon on both sides. Three days later, after being rendered toxic with salicylates, both thighs were one inch less in circumference, measured at the same level, and there was much less pain.

There have been 2 other cases less acute, but with distinct muscular swelling involving the left scapular muscles and right trapezius respectively. Also another case of indurative headache without swelling when observed, but with tenderness along the attachment of the temporal muscle.

In this connection a case may be reported from the private practice of Dr. John Phillips, with his permission. The patient was a well developed women, aged thirty years. Her general health was good and there was no alcoholic history. One year ago she had a rather mild attack of acute rheumatism with pain and

[4] Modern Clinical Medicine, Diseases of the Nervous System, p. 863.

swelling of both shoulders and a temperature of 101, associated with brachial neuritis and tenderness above the clavicle on the right side and tenderness over all the nerves of the arm. There was almost complete loss of power. She was put on oil of wintergreen until toxic. The acute pain subsided in two or three days, and the neuritis in two weeks, although it was five weeks before the strength in the arm was regained. At the same time she had severe headache and tenderness of the scalp, with thickening of the insertions of the sterno-mastoid and trapezius muscles of sufficient degree as to be discovered by the patient. This thickening disappeared under massage and salicylates in about 48 hours. Five months ago she had a similar condition of the muscles of the head with some weakness of the left arm and tenderness over the ulnar and median nerves. The headache again yielded to salicylates and massage. The neuritis cleared up in about three weeks. There was no endocarditis.

In the first 3 cases it seems highly probable that the myositis was only a part of the generalized infection of acute rheumatic fever. Of course, there is no bacteriological evidence of this fact, which would be difficult to prove, since the specificity of the micrococcus rheumaticus or any other organism is not established.

In this connection several convincing series of cases may be brought together from the literature. In 1894, Newton,[5] of Kent, reported an epidemic of 43 cases of acute muscular rheumatism seen within five weeks. The abdominal and lower intercostal muscles were most commonly involved, rendering respiration painful, short, and rapid. In some cases various muscles of the back, especially in the lumbar and sacral regions, were effected. The onset was acute, with fever of 100° to 104°; pulse, 80 to 100; furred tongue, headache, and the characteristic sour acid sweats of acute articular rheumatism. There were no nasal or pulmonary complications, which is of importance in making the differential diagnosis from influenza. Newton regarded the infection as identical with acute articular rheumatism for reasons, which he summarizes as follows: (1) There was no influenza prevalent at the time. (2) There was none of the subsequent depression characteristic of influenza. (3) They responded promptly to salicylates. (4) It was apparently mildly contagious, 3 cases occurring in one household. (5) There was endocarditis in 2 of the 43 cases; these were not treated with salicylates. (6) The epidemic occurred after an unusually wet summer and low in the valley of the Medway.

As stated before most of the reports of acute muscular rheumatism appeared in the German literature during the period of 1892 to 1897. Among the writers who expressed the opinion that the condition was the same infection as acute articular rheumatism,

[5] Brit. Med. Jour., vol. xi, p. 651.

were Sahli,[6] Rosenbach, von Leube,[7] Immermann and Saccharjin.[8] Risse[9] thought, as referred to above, that some of the cases reported as dermatomyositis were really the same thing. He reports a case in a man, aged thirty-five years, who had had acute articular rheumatism with two recurrences. His parents were also both rheumatic. The illness reported began with acute rheumatism of both ankles. After one week, the right thigh and the right tibialis anticus and peroneal muscles became tender, swollen, and indurated. Salicylates relieved the acute pain but in two days the calf and the other thigh muscles and overlying skin became tense and edematous. The testicles and abdominal wall then became similarly involved, and some hiccough occurred, suggesting slight disturbance of the diaphragm. The case was also complicated with nephritis, lasting about four weeks. Risse thought the dermatitis and edema might simply be due to a more intense or virulent rheumatic infection. He also thought that the chronic condition (*Muskelschwiele*) when not traumatic, was usually referable to acute rheumatism.

Sick[10] reported an instructive series of 9 cases occurring in epidemic form among nurses and attendants living in the basement of a hospital. These patients were well studied clinically and by blood and tissue examinations. Symptoms were initiated in some cases with sore throat, in all cases with malaise, gastric disturbances and general indisposition, followed in one to three days by rise of temperature and pain, swelling, and a diffuse or nodular infiltration of various muscles, usually of the thighs, calves, back, or arms. The skin was not affected and the lungs were clear. There were no neurological changes. There was excessive perspiration, and one case was associated with typical articular rheumatism. This case had had one year previous to the illness in question, acute pain in the right iliac region, with fever, and was operated on for appendicitis (during the febrile stage), but the appendix was found normal. Later she had joint swellings, then this epidemic muscular involvement, at which time a mitral insufficiency was observed; and finally, following this, an attack involving joints and muscles simultaneously, both of which were relieved by salicylates. All of these cases were characterized by a strong tendency to recurrence. Blood cultures were uniformly negative. There was no leukocytosis or eosinophilia. Excised portions of muscle showed no histological change, except in one case, in which there was some clouding of the structure and slight infiltration with fine fat droplets and round cells. No trichini were found. Bacterial stains of the tissues were also negative.

[6] Deutsch. Archiv. f. klin., Med., 1893, Band li.
[7] Deutsch. med. Woch., 1894, Band i.
[8] Ibid., 1894, p. 525.
[9] Ibid., 1897, p. 232.
[10] Münch. med. Woch., 1905, lii, 1092 and 1152.

Sick considered in the diagnosis here, trichinosis, dermatomyositis, Gregarinal myositis (better known to veterinary medicine), and acute rheumatic fever. The tissue examinations excluded all but dermatomyositis and rheumatic fever. Sick did not class it as a rheumatic myositis, because it did not conform to the description of that condition given by Lorenz,[11] who gives four characteristics, namely: (1) Short duration, (2) limited localization, (3) no histological changes, and (4) no infiltration of muscle. His description, however, evidently does not apply to the acute form of myositis. Although Sick classified the condition as an attenuated form of dermatomyositis, he admits the disturbing factor of an associated typical articular rheumatism in one case. The involvement of the respiratory muscles he regarded as inconsistent with muscular rheumatism, but this was a prominent feature of the cases reported by Newton, above summarized. As to dermatomyositis, the perivascular inflammation of that disease was lacking in Sick's cases.

Herz[12] also believes there were many mild cases of dermatomyositis reported at that time, which he considered identical with acute rheumatic fever. He reported 21 cases from Breslau, with 9 recoveries. Some of these cases had joint swellings, also involvement of the tendons and tendon sheaths as well as of the muscles. The skin involvement was in the form of an erythema which resembled erysipelas, and which subsided early, leaving the myositis as the prominent feature of the disease. The skin sensibility was intact, and the electrical reactions showed at most occasional diminished excitability. One of the severe cases, fatal in six days, was autopsied, and the findings reported as follows: Bronchitis and edema of lungs; a few small infarcts of the spleen, marked softening and edema of the muscles with beginning suppuration. A culture taken twelve hours post mortem showed an organism of the colon group, which Herz regarded as a secondary infection.

V. Leube[13] reported on 200 cases of muscular rheumatism from the Wurzburg Clinic. He describes its greater prevalence, in almost epidemic form, in the spring, and expresses very strongly the opinion that it is identical with acute articular rheumatism and that only the bacteriological evidence is lacking. He also speaks of the frequent muscular involvement in ordinary acute articular rheumatism, a fact which is scarcely referred to in recent textbooks. His cases usually had considerable constitutional disturbance; many, however (two-thirds of all), especially the mild ones, had no fever. He reports one-sixth of all cases as having endocarditis.

Lacquer[14] reported a case of special interest in connection with Case I reported above, from Lakeside Dispensary. It was an

[11] Jour. Exp. Med., 1898, No. 30.
[12] Deutsche. med. Woch., 1894, p. 791.
[13] Loc. cit.
[14] Deutsch. med. Woch., 1896, p. 442.

acute and recurrent myositis of the upper arm, which was incised on two occasions, three years apart, but there was no gross or histological change in the muscle or periosteum. This case later had typical acute articular rheumatism. Lacquer regarded the myositis as of the same origin, probably in attenuated form.

Kerr[15] says there is no doubt of the fact that acute rheumatic fever may have as its first manifestation, involvement of the abdominal musculature, and reports 3 cases all in children at some time subjects of acute articular rheumatism. For example, 1 case had a sudden onset of localized pain and swelling in the abdominal musculature, with temperature of 100.4°, which subsided under salicylates. Three months later the child had a typical attack of acute articular rheumatism. He is personally convinced that the two differently localized affections are parts of the same disease.

In conclusion, then, if one may accept genuine rheumatic myositis as a definite clinical entity, the question arises as to its relation to the recently described fibromyositis, indurative headache, etc. Undoubtedly the chronic nodular rheumatism (*Muskelschwiele*) of the German literature would fall under this heading. However, the recent articles of Telling,[16] and Luff,[17] etc., give it a wider application, and include various fibroid processes of the skeletal musculature. Telling does not think that it is a sequel to acute rheumatic fever, or that it occurs with any frequency in genuinely rheumatic cases. However, Stockman,[18] from whose article Telling and others quote as the most complete in the scanty literature on the subject, thought that acute rheumatism was one definite cause of the chronic form, and that the subcutaneous nodules of acute rheumatism frequently persisted as indurations or thickenings of fasciæ, nerve sheaths, etc. These thickenings he considered responsible for the neuralgic pains and even atrophy following acute rheumatism. He also suggested that the nodules may be definite localizations of the infecting organism, but that the treatment required to relieve the condition at this stage was different from that of active rheumatic fever. Miller[19] also described the subacute form of myositis with slight swelling of the muscle and pain on pressure and voluntary movement, and slight effusion into the subcutaneous tissues over the muscle after the myositis has subsided. Hence it seems probable that some cases of fibrositis may follow a previous acute rheumatic myositis, which may have been mild or unrecognized. All writers, English, German, Swedish, and French agree that massage is the only really efficient treatment. This should be carried out systematically and with the idea of dispersing the infiltrations.

[15] Loc. cit.
[16] Lancet, 1911, i, 734.
[17] Loc. cit.
[18] Edinburgh Med. Jour., vol. xv, p. 107.
[19] Trans. Med. Chir. Soc., Edinburgh, 1897–8, xvii, 1?8.

ADAMS-STOKES SYNDROME, WITH COMPLETE HEART-BLOCK AND PRACTICALLY NORMAL BUNDLE OF HIS.

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The case herewith described was first reported by Dr. Alfred Stengel and Dr. William Pepper in the Amer. Jour. Med. Sci., October, 1910, under the title, "Heart-block, with an Indication of Genuine Hemisystole." From that time onward the patient was almost constantly under observation, and many more pulse tracings were obtained before his death. An autopsy was held and a careful examination of the heart was made. The full pathological report follows later, but suffice it here to say that the bundle of His was practically normal. In addition to this interesting finding, several other features make us believe this further report on the case is fully justifiable.

Six cases have been previously reported of heart-block without any lesion of the bundle of His, or with but such slight alteration of the bundle, as might be expected in many hearts where no block had occurred.

Krumbhaar,[1] in 1910, in his article on "Adams-Stokes Syndrome, with Complete Heart-block, without Destruction of the Bundle of His," states that he could find but 2 cases similar to his own, and since that time we have been able to find but 3 additional cases.

Deneke and Fahr's[2] Case. This case was one of complete block. The authors admit that they did not succeed in completely examining the bundle which was abnormally long but found no lesion in the parts examined.

Nagayo's[3] Case. Here the heart-block was only incomplete. No lesion of the bundle was found, but there was extensive myocarditis, and Nagayo believes that the block occurred through the altered musculature of the ventricle.

[1] Arch. of Int. Med., 1910, v, 583.
[2] Arch. f. klin. Med., 1906, lxxxix, 39. See also Virchow's Arch., 1907, clxxxviii 562.
[3] Zeit. f. klin. Med., 1909, lxvii, 495.

KRUMBHAAR'S[4] CASE. Here complete heart-block with Adams-Stokes syndrome was known to have existed for five years. At autopsy a chronic myocarditis of the ventricles was found. The bundle of His was traced in unbroken continuity from Tawara's node to beyond the bifurcation, with no abnormality other than a slight increase in connective tissue, no greater than is found in other hearts that have never exhibited heart-block. Fibrous changes were found in the muscle bundle of Keith in the sino-auricular junction.

GRIFFITH AND COHN'S[5] CASE. This is a case with lengthened *a-c* interval and with attacks of partial and complete heart-block, and showed the bundle of His continuous from the auriculo-ventricular note to the bifurcation, although its diameter was somewhat reduced and the fibers compressed by diffuse fibrosis. Beyond the bifurcation it was interrupted by granulation tissue.

MALLARD, DUMAS, and REBATTU'S[6] CASE. This was a case of partial block when tracings were made, but in which probably complete block had occurred at times, as spells of unconsciousness are mentioned. At autopsy there was an extensive fibrous mediastinitis, with involvement of the vagi. The region of the bundle of His was sectioned and but one in twenty of the sections stained. No lesion was demonstrated.

HOLST AND MONRAD-KROHN.[7] The authors report a case of a male, aged fifty-four years, who for eight days had repeated attacks of syncope, with disappearance of the radial pulse. Between the attacks the pulse varied from 40 to 80, Tracings showed at times a partial block. At other times a slowing of both auricle and ventricle. At autopsy no lesion of the a.-v. bundle was found, except that it was slightly more fatty than usual. Both vagi, however, showed evidence of extensive degenerative neuritis. The authors believe the cardiac attacks to be of vagal origin.

AUTHOR'S CASE.

The following chronological list of events will serve graphically to place the patient's life on view:

1871, born in Italy. 1894, syphilis, in Philadelphia Hospital. 1900, married. 1902, wife aborted. 1903, child born, died from burns. 1905, child born, living and well. 1906, child born, living and well. 1907, child born, living and well. 1907, patient had attacks of epigastric pain. 1908, child born, living and well. Patient fainted. 1909, severe pains and syncopal attacks. January 25, 1910, pains. February 5, admitted to University Hospital,

[4] Loc. cit.
[5] Quart. Jour. Med., 1909-10, iii, 126.
[6] Arch. des Maladies du cœur, Paris, 1911, iv, 298.
[7] Quat. Jour. Med., 1911, iv, 498.

with complete heart-block. February 8, 1.30 P.M., complete heart-block still persisting; patient given two hypodermics of atropine. 5.30 P.M., incomplete block 3-1 rhythm. February 9, incomplete block 2-1 rhythm. February 10, 2-1 rhythm, alternating with normal rhythm. February 24, left University Hospital, not having had while there a single syncopal attack. March to May, seen at office, either 2-1 rhythm or normal rhythm. In May started to work as cabinetmaker again. May to October, worked. October 22, slight syncopal attack. October 25, syncopal attack, and patient was taken to Jefferson Hospital. October 26, had six to eight syncopal attacks. October 27, had four or five attacks. Refused to stay longer in hospital, and went home. October 28, one attack; admitted again to Jefferson Hospital; discharged next day. October 30, had four syncopal attacks. December 5, the patient came to the University Hospital to see Dr. Pepper, who found him sitting in the waiting room. The man stood up, and told Dr. Pepper how glad he was to see him again. He suddenly grew pale, put his hand to his heart, fell over on a bench, and had a general convulsive attack, with unconsciousness. He had at first stertorous breathing followed by shallow breathing. In about a minute his radial pulse, which had been absent, returned and immediately the color came back to his face and he regained consciousness, and in a few minutes walked up to the ward, having been persuaded to stay for a while in the hospital. This was the first time we had seen him have a syncopal attack. His radial pulse on this second admission to Dr. Stengel's wards was 44, falling shortly to between 24 and 36.

December 6. Tracings showed complete block, or complete dissociation of auricular and ventricular action, and during the patient's stay in the hospital this persisted unfailingly. There were no syncopal attacks on this day. Atropine, which had during his first stay in the hospital apparently interrupted the complete block, changing it first to partial block and then to normal rhythm, had no beneficial effect.

December 7. The following notes were made by the resident physician, Dr. Sledge: Patient's pulse has continued to run at about the same rate. This morning he had an attack, and was watched by one of the nurses. His pulse stopped for a few seconds; respirations at first were slow and noisy; face remained flushed, and consciousness was not lost. Pulse returned rapid and fluttering, respirations came back to normal, and in two minutes pulse had slowed down to 20. Patient was much exhausted. At 11.30 today, while sitting down on patient's bed to take his blood pressure, he looked up at me and said, "I think I am going to have another spell." I reached for his wrist and felt only one beat of his pulse; he became very pallid, there was a general muscular spasm with twitching of his limbs, followed in less than a minute by opisthot-

onos. (His opisthotonos lasted about a minute, followed by relaxation and intermittent spasms of extremities; absolute relaxation occurred when respirations stopped). No heart sounds were audible from cessation of pulse to its return; after that they were distinct. Breathing became rapid at first and very noisy, beginning to become slower and less noisy at the expiration of one minute, stopping altogether at the end of four minutes from the beginning of his attack. After respiration stopped, the patient slowly became cyanosed (he had been pallid before), and the vessels of the neck were distended, and no venous pulsation could be detected; he lay in this condition for nearly three minutes, and was apparently dead. Artificial respiration was begun, with head hanging over side of bed, and with my finger over his radial artery; at the expiration of one minute, his radial pulse suddenly recurred, soft and running, and his face became very much flushed; artificial respirations were stopped and he continued to breathe, respirations being slow and shallow. In less than a minute he was perfectly conscious, and attempted to move in bed; pulse had slowed down to 20. On being asked how he felt, patient said that he was thoroughly exhausted. Flushing was quickly followed by pallor. I asked the patient why he told me that he thought that an attack was coming on, and he replied that everything had begun to appear black, and his vision was disappearing. One hour after the attack was over the patient expressed a wish to die, and said such an end could not be far off. The thought of these attacks is a great mental strain on him. Patient had another attack tonight at 9.45; pulse stopped for over three minutes; respirations did not cease, and recovery was very rapid.

December 8. Patient had another attack at 12.30 last night, with loss of consciousness, but no cessation of respiration; attention was called to him by his noisy breathing. Four minutes after the nurse reached his side the radial pulse returned. After the attack had subsided, the patient was very hysterical for half an hour, believing that he was near death. He was quieted by a small dose of morphine. Patient continued restless and nervous the rest of the night; this was especially marked at six, and again at eight o'clock this morning; at both of these times he thought an attack was coming on, but pulse did not stop, nor had he respiratory difficulty. At 12.30 today patient was given an injection of "606" by Dr. Longcope. Dr. Pepper made pulse tracings before and after the injection, but no change was noted. At 5.15 tonight patient had some respiratory difficulty for a few seconds; pulse did not stop, nor was consciousness lost.

December 9. Patient had a short attack this morning at two o'clock; another attack came on at 8.30, and lasted over four minutes from the time the nurse reached his side; artificial respiration was required. At 11.30 he had another attack that lasted six minutes; respirations and jugular pulse stopped; eyes showed mark-

edly dilated pupils; after this he was wildly hysterical for a half hour. Another attack occurred at 4.30 and lasted about six minutes; artificial respiration was required for nearly a minute; violent mania was present after return of pulse for fifteen minutes. Patient was taken home in the ambulance at his own request.

He had on arriving at his home another severe attack, while the resident physician who accompanied him was present, and then for seven weeks was entirely free from them, although we had told him that he would undoubtedly have them and that he would die within twenty-four hours after leaving the hospital. He was visited at his home every two or three days for seven weeks, tracings being made on each occasion by Dr. Pepper. On December 13, 1910, he showed an incomplete block with a 3-1 rhythm, and on December 22, 1910, an incomplete block with a 2-1 rhythm; but the rhythm never became normal, and with the exception of these two occasions there was always complete auriculoventricular dissociation. Just before Christmas, 1910, his wife gave birth to a healthy boy; but this excitement in the family brought on no syncopal attacks in our patient. This child showed a negative Wassermann, as did its mother, although the patient on two occasions had given a positive reaction himself. He remained in the house sleeping on a lounge and walking around the first floor, but not venturing on the street, and on February 1, 1911, having been much wrought up over the withdrawal of his weekly income from an insurance company he had three syncopal attacks in one day and died during the last one.

A partial autopsy was performed by Dr. Austin about eight hours after death, and the heart removed. It was found to be greatly distended with dark fluid blood, a large quantity running out on cutting the vessels. The right side was particularly distended, the auricles, although enlarged, were not so extremely distended in proportion to the ventricles. The valves were perfectly healthy, and no macroscopic change in the heart, aside from the dilatation, was found. During life the right border was more displaced than the left, and there had been always noted a long systolic murmur at the apex. The patient's fingers had become quite clubbed, and the nails curved during the last year.

Autopsy Protocol. Subject is a fairly well-developed Italian, male, adult. Little panniculus adiposis is present; no edema; no scars. An incision was made along the middle of the sternum, the skin and muscle reflected, and the sternum removed in the usual manner. The abdominal viscera were not fully exposed, owing to restrictions under which the autopsy was conducted.

The pleural cavities were free from fluid; the lungs appeared normal. The pericardium was much distended, and on being opened contained 30 c.c. of clear fluid and a greatly distended heart. The cardiac distention was most marked on the right side.

On cutting through the vena cavæ the entire mediastinum filled quickly with fluid blood. The heart and four inches of the aorta were removed for study. Sections were taken for histological study from the liver and from the right kidney, which was lobulated and deeply congested.

The heart weighed 420 grams; epicardium was smooth and glistening; moderate amount of subepicardial fat. Blood in the cavities was almost entirely fluid. The myocardium was grossly normal. The left ventricular wall at the base was 14 mm. and the right ventricular wall 3 mm. thick. The endocardium was smooth, no lesion being visible in the region of the bundle of His or of the sino-auricular bundle. The valve orifices measured in circumference: tricuspid, 15 cm.; mitral, 10 cm.; pulmonic, 8 cm.; aortic, 6.5 cm. Valve leaflets were thin and pliable. The region of the sino-auricular and His bundles was preserved intact for serial sectioning. Sections were taken also from other portions of the myocardium. The aorta showed a few nodules apparently beneath an intact intima.

Histological Protocol. Autopsy No. 3559, '99, 12. Report by Dr. Austin. The following tissues were prepared and submitted for histological examination: Heart, aorta, liver, kidney.

Heart: Sections through myocardium and endocardium show ventricular muscle fibers well developed. Nuclei stain normally. Striations are distinct. Throughout the sections there is a moderate diffuse fibrosis, which around some of the larger vessels is quite marked. The arteries show thickening of their adventitia. The endocardium is normal. The vessels are moderately filled with blood.

Sino-auricular bundle of Keith-Flack: Several transverse sections were made through this bundle and stained with hematoxylin and eosin and with Mallory's connective-tissue stain. The muscle fibers of the bundle appear well developed. There is conconsiderable interstitial tissue in the bundle, not, however, an amount that can be considered excessive, the connective tissue forming a considerably less conspicuous feature of the cross-section than does the muscle tissue.

The bundle of His: The bundle was examined by serial longitudinal sections and stained with hematoxylin and eosin and with Mallory's connective-tissue stain. The course of the bundle was followed from Tawara's node to just beyond its bifurcation as an unbroken bundle, about 5 mm. in width. The course of the bundle is somewhat S-shaped. The muscle fibers of the bundle are slender and very closely approximated, with little interstitial tissue. The nuclei stain well. The cytoplasm shows distinct striation.

Aorta: Section shows nodular fibrous thickening of the deeper portion of the intima and fibrosis of the inner layers of the media. Musculature of the remainder of the media is well preserved.

Liver: Section shows a normal capsule. The lobules are distinctly demarcated. Immediately around the bile ducts there is a moderate fibrosis, with round-cell infiltration. The bile ducts show normal mucosa. The parenchymatous cells are small and well defined. Their nuclei stain well. In a few of these cells one or two small fat vacuoles are seen. Throughout the lobules the cells show a moderate deposit of intracellular, pale yellow, amorphous pigment. The capillaries and the larger vessels contain little blood.

Kidney: Section shows a thin capsule. The glomerular tufts are large filling the capsules, and show moderate congestion; otherwise normal. Tubular epithelium shows well defined finely granular cells; nuclei are well stained. In a few of the convoluted tubules the epithelial cells appear moderately swollen. In some of the lumens is a little granular or homogeneous material. The tubules are in close apposition and there is but little interstitial tissue. Throughout cortex and medulla is marked congestion, without pigmentation. Treponema pallidum could not be demonstrated in the liver, kidney, or aorta by the Levaditi method.

CONCLUSIONS. The special points of interest in this case, in addition to those noted in the first report,[8] that we wish to bring out in this paper are:

1. That we have here a progressing typical case of heart-block lasting for three or four years, at first partial block alternating with normal rhythm, with occasional attacks of complete block, and later persistent complete block, and yet at the autopsy apparently no sufficient lesion was discovered in the bundle of His to explain the block.

2. The giving of a dose of "606" to the patient while he had complete heart-block and was having attacks daily of Stokes-Adams syndrome without any noticeable good or bad result.

3. We wish to call attention to the extreme length of several of the syncopal attacks. One attack timed by Dr. Sledge, the resident physician, lasted eight minutes, during the last four minutes of which the auricles apparently stopped beating in addition to the cessation of the ventricles. Apparently the man was dead, but artificial respiration brought him to life. Another attack witnessed by Drs. Edsall, Longcope, Sledge, and Pepper, and a number of medical students, was nearly as long, and very dramatic. This lasted six minutes, and for fully three minutes the auricles had apparently stopped beating, as no sounds could be heard over the chest, no pulsation noted in the neck, no respiratory movements occurred, and the patient's color, which at first had been white, became dusky. We all thought the man dead except Dr. Sledge, who, with some assurance on account of his previous

[8] AMER. JOUR. MED. SCI., October, 1910.

success, began artificial respiration, and again restored the man to life and consciousness, which was promptly followed by a violent hysterical outburst, in which the man sang, cried, and shouted, and though none of us understood much Italian, we frequently heard the words "La morte" uttered with the most blood-curdling clearness. That these periods of apparent cessation of auricular action may have been due to auricular fibrillation cannot be denied.

4. In the first report of this case, already referred to, attention was particularly called to what was believed to be a genuine instance, though an isolated single one, of hemisystole. Apparently, as shown by the tracing on a single occasion, the left ventricle did contract normally after the auricular contraction; but the right ventricle did not contract, or possibly contracted very feebly, and the following statement was made in that article: "Probably it would be unwise, from this single observation, to say that in this particular case the division of the bundle of His going to the right ventricle was more involved or damaged than the division going to the left ventricle; but if further similar findings had been noted possibly such a localization could have been made."

The serial sections showed at the bifurcation of the bundle the branch passing to the right ventricle slightly encroached upon by an area of fat and fibrous tissue, while the branch to the left ventricle was normal.

It is, of course, difficult if not really impossible to properly interpret the concurrence of the single hemisystole and the slight lesion of the right branch of the bundle of His, if lesion it was. No other hemisystoles were noted, and the slight encroachment on the right branch of the bundle was not more than is sometimes found in apparently normal hearts.

5. It is interesting to note the occurrence of clubbed fingers in this case without any valvular disease of the heart or pulmonary disease.

6. That although during the man's first stay in the University Hospital he showed for several days constant irregular ventricular action during complete block without syncopal attacks, he never showed this irregularity later, even though he had syncopal attacks and complete block.

CLINICAL OBSERVATIONS OF REFLEX VAGUS PHENOMENA GROUPED IN SYMPTOM COMPLEXES.[1]

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It is a well-known physiological fact that nerve impulses may reflexly produce the most diverse results, such as glandular secretion, regulation of blood supply, sweating, and muscular contraction; yet the role that nerve irritations and abnormal nerve impulses play in the production of reflex symptoms and the significance of such impulses in the elucidation of symptom complexes has not received the attention its importance warrants. In this paper I shall deal only with so-called functional disturbances of vagus impulses, both excitor and inhibitory in character, and shall exclude all organic affections that influence the pneumogastric. Before proceeding with the study of the clinical cases of vagus excitation, I shall briefly survey some facts in connection with the significance of the vagus in certain symptom complexes, and offer some general considerations on the subject of reflexes and the radiation of impulses.

Von Noorden[2] was the first to group symptoms resulting from irritations of the vagus. He confined his observations to purely hysterical cases ("hysterical vagoneuroses") presenting heart irregularities and hyperesthesia or anesthesia of throat and larynx. In describing a number of cases, he stated "if for any reason (organic stomach disease or nervous dyspepsia) hysterical people have gastric symptoms, functional disturbances of the vagi are common." Gowers[3] described "vagal attacks" which consist mainly of subjective symptoms, gastric, respiratory, cardiac, or vasomotor in nature, the latter causing constriction of vessels and coldness particularly of the extremities. He believed these attacks usually occurred as epileptic equivalents.

Meltzer[4] has done pioneer work in this country in directing attention to the clinical importance of the inhibitory nerves in both normal and pathological states; he ascribed several diseases of clinical importance (for example, cardiospasm, "icterus emotif") to disturbance of the function of inhibition. He believes that "an impulse which causes inhibition in a centre extending to a special function irradiates, spreads from there to other centres, and functions neither automatically nor physiologically connected with the primary centre." To quote from another part of the communication by the same author: "We learn that between the antagonists

[1] Read at the meeting of the Harlem Medical Association, New York, December 6, 1911.
[2] Charité Annalen, xviii, 249. [3] Grenzgebiete der Epilepsie, etc., 1908.
[4] New York Med. Jour., May 13, 20, 27, 1899; Medical Record, June 7, 1902.

(vagus and sympathetic) it is often practically the effect of only one force which is manifest during stimulation, and that it is neither always inhibition nor always excitation which is more favored by the stimulus."

It is, however, mainly due to the recent excellent clinical and experimental studies of Eppinger and Hess[5] upon the condition they have termed "vagotonia" that distinct progress in the clinical study of vagus excitation and inhibition has been made. They have borrowed the term "vagal tone or vagotonia" from experimental physiology, where it has long been used, and have applied it clinically to those in whom the vagus system is particularly irritable and susceptible to stimulation and inhibition, and in whom certain vagotropic drugs, mainly atropine and pilocarpine, have respectively marked inhibitory and excitive (tonic) effects.

After dividing the nervous system into the animal and vegetative, Eppinger and Hess subdivide the vegetative into the sympathetic and the autonomous or system of the "enlarged vagus" ("erweiterte vagus"). Sympathetic and vagus nerve actions are antagonistic, the autonomous vagus impulses tending not only to antagonize but to partially paralyze the former. In connection with the cases to be described, it is of interest that these observers indicate that vagus irritations may produce active gastric peristalsis and even vomiting, and that the crises of many infectious diseases are coincident with profuse perspiration and cold extremities—both due to irregular blood distribution from disturbamce of the vasomotor system. In many instances the above authors have succeeded in differentiating pharmacologically the two systems, sympathetic and vagus; for example, adrenalin irritates and excites only the sympathetic system, while atropine, pilocarpine, physostigmine, and muscarine have an elective action on the vagus system. Of these latter drugs, atropine often nullifies the effects of vagus stimulation, and hence partly antagonizes the action of muscarine, pilocarpine, and physostigmine.

A word in reference to the spread or radiation of impulses from one centre to another. Mackenzie[6] states that abnormal stimuli of the sympathetic centres in the cord may spread beyond it and affect cells in the immediate neighborhood. Furthermore, according to Nagel[7] there exists spinal as well as central impulse radiation. He quotes Heads' theory of spinal segmentation in skin hyperesthesia to prove spinal radiation of impulses.

I shall employ the term vagus excitation in the study of my cases, because, except for the marked salutary effect of atropine and the extreme vagus irritability in illness, the patients did not show when

[5] Zeit. f. klin. Med., 1909, vi and vii, 345; lxviii, 205, Sammlung klinische Abhandlungen über Pathologie und Therapie des Stoffwechsels und Ernährungsstörungen, p. 9.

[6] Diseases of the Heart, second edition, p. 33.

[7] Handbuch der Physiologie des Menschen, iv, 290.

normal any of the signs of "vagotonic disposition" described by Eppinger and Hess, namely, a tendency to perspiration, flushed face, moist skin, enlarged tonsils, slow pulse, frequent eructations, and generally increased reflexes. It may be that in some individuals the "vagotonic disposition" is not evidenced until strong abnormal excitation stimuli are present.

Some of the patients to be presented were "neurotic" in the ordinary acceptance of the term; in not a single instance, however, were there any hysterical stigmata.

Before proceeding with the clinical presentation, I wish to devote a few lines to the anatomy and physiology of the pneumogastric. The vagi originate in the nuclei in the lower part of the floor of the fourth ventricle, leave the skull through the jugular foramen and descend vertically to the root of the neck. Each vagus then descends along the esophagus, perforates the diaphragm, and spreads over the surface of the stomach. Filaments from the latter portion of the nerve anastomose with the solar, celiac, splenic, and hepatic plexuses of the sympathetic system. In the neck the main branches of the vagi supply the pharynx, esophagus, and larynx. In the thorax, the main branches are those that help to form the pulmonary and the cardiac plexuses. The cardiac branches anastomose[8] with the cardiac branches of the sympathetic and form the deep and superficial cardiac plexuses inervating the heart.

According to the most recent studies, cardiac tissue possesses the functions of rhythmicity, excitability, conductivity, contractility, and probably tonicity. Most of these functions are profoundly influenced and inhibited by vagal stimulation.[9] Upon the distal ends of the pneumogastric, peripheral stimulation of the corresponding branches of the vagus produces increased salivation (chorda tympani), contraction of pharynx and cardia, "gastric hypersecretion with or without hyperacidity, increased violent gastric peristalsis, which may easily lead to atypical (retrograde) peristalsis and even vomiting." (Eppinger and Hess.) Regarding the nervous mechanism and cause of peristalsis in the small intestine, the effect of the vagus is complicated not only by the action of the sympathetic (splanchnic nerves), but also by the automatic action of the Auerbach and Meissner plexuses. It is definitely known, however, that increased vagus excitability may give rise to abnormal tonic peristalsis of the small intestine. I shall not further enter into the effects of vagus stimulation on the musculature of the gall-bladder, on the pancreatic ducts, the lower colon and rectum, the genital organs and the blood, except to state that the vagus also affects all these organs, and in the blood, according to Eppinger and Hess, produces eosinophilia.

[8] Dogiel-Pflueger's Arch. f. gesammte Physiologie, September, 1911.

[9] Gaskell, Jour. Phys., 1886, vii, 1; Mackenzie, Diseases of the Heart, 1908, p. 7; Nagel, Handbuch der Physiologie des Menschen, i, 260.

From the above we observe that throughout its long course the vagus sends branches to many important structures and is dircetly concerned with many vital processes; *a priore*, then, it can be readily seen that abnormal peripheral irritation in one end organ may produce diverse reflex results not only in adjoining, but also in remote parts of the body.

I now wish to describe a series of cases, eight in all, which, of many that I have seen, have particularly and strikingly impressed me as showing the importance of abnormal irritation of the vagus in various clinical conditions. Some of these observations were made in patients with organs already the seat of pathological change; in these the added vagus irritation had a particularly marked effect upon the symptoms present.

CASE I.—W. D., aged twelve years, a strong, healthy boy. A general physical examination made several weeks before his present illness, was entirely negative. The boy's illness consisted of an attack of influenza (bronchitis) with two relapses (mild coryza, mild eustachitis). Each attack began with a temperature of 102°, lasted about four days, and ended in a mild crisis, accompanied by vomiting, sweating, and an irregular pulse. The pulse dropped from 80 to 50, caused by varyingly long pauses between the beats both at apex and wrist. No polygraphic tracings were made so that the exact nature of the irregularity could not be determined; it was probably, however, of the type described by Mackenzie[10] as sinus irregularity and commonly ascribed to the effect of vagus inhibition on the normal rhythm. There was absolutely no sign of any heart disease present—no decompensation, no murmurs, no dyspnea, no edema. Except at the time of the crisis, the patient felt absolutely well.

The features of this case which point toward vagal influence are the events occurring at the crisis: (1) The irregular pulse; (2) the vomiting; (3) the sweats.

1. *Irregular Pulse.* The influence of the vagus on the normal sinus rhythm has already been mentioned. An interesting phenomenon in conjunction with the irregularity was that forced rapid breathing a few times always overcame the sinus irregularity; the pulse became regular and remained so for a few minutes. The explanation of this phenomenon appears to me to be as follows: normal rapid impulses originating in the vagal pulmonary plexuses temporarily overcame the disturbing effect of the influenza toxin on the vagus centre and thus temporarily produced a rhythmical pulse.

2. *Vomiting.* It is well known that vomiting can be readily produced by central vagus disturbance. In this patient the toxemia

[10] Diseases of the Heart, 2d ed., p. 141.

acted upon the vagus centre and, irritating the gastric filaments, caused peristalsis and emesis.

3. *Sweating.* This is, of course, an almost invariable symptom of fever crises. It is here mentioned on account of its connection with the symptoms of vagus excitation. Because of the effect of atropine (vagotropic), Eppinger and Hess have determined, that despite generally accepted physiological and anatomical conclusions, sweating is under the influence of the vagus system and not of the sympathetic. It appears to me, however, that the critical sweats are due to the radiation of the impulses from the vagus centre to the sympathetic system.

Crises such as above described are quite common, particularly in febrile affections, but sufficient emphasis has not been placed upon their cause. I would suggest the name vago-excitor crisis for such a symptom-complex. Further reference will subsequently be made to this condition.

Case II.—Mrs. F., aged sixty years; myocarditis and marked arteriosclerosis. In June, 1910, she had an influenzal pneumonia that involved three different lobes of the lung (pneumonia migrans). Each pneumonic involvement, lasting five days, ended in a very sharp crisis: extreme prostration, sweating, severe retching and vomiting, and extremely irregular and tumultuous heart action (so-called delirium cordis). The irregularity consisted in complete arrhythmia, the beats followed no rhythm, the rate varied momentarily between 60 and 160, strong and weak impulses following each other in a haphazard manner.

Despite arrhythmia and the pulmonary involvement there was no dyspnea and no pulmonary edema. Between the crises and during the course of the pneumonia the pulse was usually regular and of good quality. A distinct herpes labialis et nasalis accompanied each crisis. After the pneumonia there was, for some weeks, edema of the legs and an occasionally irregular pulse.

Case III.—G. R., aged forty-three years, nephew of Case II, had never been previously ill. In 1910 he had a grippe pneumonia, involving in succession five different areas of the lung (pneumonia migrans). The breathing remained 28 to 36 per minute throughout his illness. At the end of each pneumonic process there was a crisis, with sweating and nausea, the temperature dropped from 105° to 100½°. With each new pneumonic involvement, and usually two to four hours before audible physical signs were present, the pulse became exceedingly irregular, exactly as in Case II. With it all the patient was conscious: there were no cold extremities, no dyspnea, no edema of the lungs, no decompensation. When physical signs of a new involvement became evident the cardiac irregularity disappeared. The patient entirely recovered and no signs of any heart irregularity remained.

Grouping Cases II and III, we again have examples of marked

vago-excitor crises, the influenza toxin—more severe than in Case I—not only inhibiting heart action, but temporarily paralyzing the vagus centre, thus producing very pronounced arrhythmia.

It appears illogical to me to ascribe the cardiac irregularities accompanying in the one case each pulmonary crisis, and in the other, each new pulmonary involvement, to a fresh myocarditis lasting from minutes to hours and then disappearing, leaving no permanent trace. On the other hand, it is well known that toxemias do involve nerve structures and end organs. Furthermore, the more prominent symptoms of acute myocarditis—dyspnea, edema of the lungs, cyanosis, etc.—were absent, despite the active and widespread pneumonia.

Despite their apparent gravity, conceiving the cardiac irregularities in these three cases as part of a vagus symptom-complex and mainly functional, I was able to give a good prognosis in each case.

CASE IV.—Mrs. H., aged fifty years; is passing through the menopause. Except for "rheumatism" of the legs, she had always been well. Three years ago, without other apparent cause, twenty-four hours after a death in the family, she suddenly developed extremely severe epigastric pains and sensitiveness; with exacerbations and remissions, this pain lasted for months. There were oft-repeated hunger pains both day and night. Acids even in minute amounts increased the gastric distress. There was no difficulty in swallowing. Abdominal examination showed the stomach outline to be small; at times the whole epigastrium felt as if it were occupied by a smooth, globular, semisolid mass. At such times, though abdominal pressure was painful, it was immediately accompanied by loud, explosive, continued eructations of gas having no odor or taste; these eructations sometimes lasted a half hour and were followed by temporary relief from pain. Repeated observation shows that there is apparently no air swallowing. After six months of the above symptoms, examinations of the fasting stomach showed no gastric juice or stagnant stomach contents. Upon one examination there was fresh blood on the end of the stomach tube. One-half hour after an Ewald test meal, very little stomach contents remained: examination showed it to be well digested: total acidity, 70; free HCl, 25; no lactic acid. After three days of meat-free diet there was no occult blood in the feces. During the next few months the symptoms were somewhat different in character. There were occasional acid eructations and vomiting of sour fluids in the morning. The patient complained of boring pains high up in the epigastrium, increased on pressure. There was a marked zone of skin hyperesthesia (Head's zone) corresponding to the stomach; this zone was present for a number of weeks. Several times, despite extreme care in diet, there were very sudden explosive attacks of diarrhea and vomiting. The patient frequently had cold feet and

cold, blanched hands. She also complained of palpitation, though the heart beat was not rapid nor irregular. During one year the patient lost forty pounds in weight. Under the routine treatment for ulcer (rest in bed, alkalies, diet, etc.) and the addition of atropine, the patient gradually improved. Since the above symptoms subsided the patient has occasional attacks of mild epigastric distress and eructations. There has been a gradual incresae in weight.

The salient features of this case are: (1) Acute onset after shock; (2) spasmodic contractions of stomach wall and disturbed gastric secretion; (3) after some months, the probable development of an ulcer. The details of this case will be discussed in conjunction with the following:

CASE V.[11]—Mrs. C., aged forty-four years, mother of three children, had always been absolutely well. Ten years ago she witnessed an accident—a man fell from a window and was killed before her eyes. She was profoundly shocked by the accident, and that *same night* developed severe epigastric pains. The patient remained in bed for six weeks, suffering from continued, intense boring pains in the epigastrium radiating to the spine between the angles of the scapulæ, vomiting of sour fluids and masses, retching and palpitation accompanied by a tingling sensation in the seventh left interspace. These symptoms were present by night as well as by day. Since that time, during all these years, the patient's symptoms have continued, so that for periods of weeks, when pains or vomiting were particularly severe, she had to remain in bed. In addition to the initial symptoms, there were frequent attacks of very severe epigastric pains, followed and temporarily relieved by loud, explosive eructations lasting several minutes; of sudden choking sensations, during which she could swallow only fluids; of frequent dizziness; of attacks of constipation, in which the bowels moved only once a week; of marked epigastric hyperesthesia. The patient lost 35 pounds in weight; she subsisted mainly upon bread and butter. One year ago the gastric symptoms—nausea, vomiting, and pain—again became acute. The diagnosis of gastric ulcer and pyloric stenosis was made. She was operated upon, the ulcer excised and a posterior gastro-enterostomy was done.

Immediate post-operative course. The temperature was normal throughout, there were no signs of peritonitis or wound infection. Her bowels moved the second day after operation and daily thereafter. For one week there was constant intractable vomiting and hiccough, the pulse was irregular and dropped to 50. Finally the stomach became more tolerant, the vomiting and singultus ceased, the pulse became regular. After one year the patient gained 15 pounds, and at the present time has excellent appetite and digestion.

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The important features of this case are : (1) Onset immediately after shock; (2) gradual development of a gastric ulcer; (3) postoperative symptoms due to exceedingly powerful vagus and phrenic excitation.

Before proceeding with an analysis of Cases IV and V, we must for a moment digress to briefly consider the important theories and experimental work on the etiology of gastric ulcer.

Vagotomy[12] has been the operation most recently employed in the experimental production of gastric ulcer. Kawamura reviewed the work in this field, performed a considerable number of vagotomies in rabbits and dogs, and has made a careful study of the pathological findings and of the stomach contents in his series of cases. Although his results are by no means uniform, it may be definitely stated that in many rabbits (27 out of 37) gastric ulcer followed the division of the vagi.

The theories that have been held upon the etiology of ulcer are varied. Virchow believed the cause to be local circulatory disturbances, thromboses, or hemorrhagic erosions, the normal neutralization of the acid gastric juice by the blood not taking place. According to Payr,[13] retrograde hemorrhagic infarcts are responsible for the fundamental changes that lead to ulcer. Diminished alkalinity of the blood (Pavy), compression of the mucosa in the folds of the gastric mucous membrane with resultant erosions are other theories. Grouping more recent opinions Ewald[14] states that the most important factor is the hyperacid gastric juice acting upon circumscribed areas of altered mucosa (of mechanical, chemical, thermal, vascular or infectious origin). Thus the fundamental cause of gastric ulcer remains unknown.

Returning to a consideration of Cases IV and V, it appears to me that these two instances conclusively prove that intense functional nervous influences—shock—may, by powerful excitation of the vagi, produce a primary gastric neurosis and consequent disturbances of gastric secretion, and possibly, also, produce disturbed gastric circulation, and finally result in the formation of an ulcer.[15] The acute gastric symptoms immediately followed the mental shock which our two patients underwent; in fact, the march of symptoms dated from the moment of the psychical disturbance. In addition, Case V indicates well an extreme sensitiveness to vagus influences. Persistent vomiting and disturbed heart action (slow pulse) followed the operation of excision of the ulcer. It appears to me that these symptoms were due to the irritation of the gastric vagus branches from the necessary manipulations of the operation as well as to the

[12] Zironi, La Riforma Medicale, 1906, xv, 395; Von Yzeren, Zeit. f. klin. Med., 1901, xliii, 181; Gibelli, Archives Internationales de Chirurgie, 1908, iv, fasc. 2.; Donati, Centralblatt f. Chirurgie, 1904, p. 346; Kawamura, Deut. Zeit. f. Chir., cix, H. 5, 6, May, 1911.

[13] Arch. f. klin. Chir., 1907, lxxxiv, 799.

[14] Modern Clinical Medicine, 1906.

[15] Stockton New York Med. Jour., October 30, 1909.

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The pronounced effect of atropine in Case IV is of interest. Although atropine has been administered to this patient for more than a year, very minute doses ($\frac{1}{800}$ of a grain) continue to control the gastric symptoms.

Case VI.—Mrs. B., aged eighty years. She is quite stout and has a slight fatty myocarditis; no apparent arteriosclerosis. One year ago she suffered from left-sided kidney colic and pyelitis. After several days a stone was passed. The pyelitis, accompanied by remissions and temperatures, lasted over two months. Soon after the fever subsided there developed severe bilateral intercostal neuritis, with extreme hyperesthesia in the corresponding intercostal spaces at the intercostal nerve exits. During the attacks of neuritis (which lasted intermittently for months) one or more of the following symptoms suddenly appeared: choking sensations, pains in the gums, spasmodic cough, either during the day or at night, difficulty (though no pain) in swallowing, sudden vomiting of solid and liquid food often immediately after they were swallowed, expectoration of large quantities of pure saliva occasionally awakening her from sleep. These different attacks were usually accompanied by exacerbations of the neuritis. Throat, esophagus, lungs, and kidneys were normal. Except for the neuralgic intercostal pain and the attacks above described the patient was in excellent general health.

That the intercostal neuritis was due to the septic pyelitis is evident, and it appears unquestionable to me that abnormal impulses from the intercostal nerves, passing through the spinal cord, affected the vagus centre and thereby caused the various vagal irritative symptoms described—laryngeal, pharyngeal, esophageal and gastric spasms, and salivary hypersecretion.

Case VII.—Mrs. R., aged sixty-nine years, has marked arteriosclerosis, myocarditis, and splanchnoptosis. Except when ill from any cause, her pulse is regular. Two years ago, after a vaginal hysterectomy for uterine tumor, there were several severe attacks of vomiting, with irregular heart action. About one year ago, following worry over the illness of her son, she had a very sharp attack of acute gastric dilatation. The temperature was 100.4°. The pulse and heart, until that time only occasionally arrhythmic, now became very irregular. There was no dyspnea, no decompensation. With lessening of the gastric symptoms the heart became more regular and only occasionally intermittent. Two months after the attack the heart was still irregular, and for the first time there were signs of heart failure: dyspnea, cough, and slight edema of the legs. Examination of the arterial and venous pulse by means of polygraphic tracings shows so-called complete irregularity of the

pulse (auricular fibrillation). Following the attack, the patient suffered for months from intercostal and abdominal neuralgias, with superficial and deep hyperesthesia, in the corresponding regions. There was also one very severe attack of constant belching, lasting about one half hour without any apparent cause.

Comparison with Case VI shows the reflex arc through the spinal cord exactly reversed. Here vagus excitation resulting from acute gastric dilatation affected not only the heart, but also reflexly the intercostal and abdominal nerves, with resultant neuralgias. The belching was due to sudden excitation of the gastric vagus filaments, producing spasmodic contraction of the stomach.

CASE VIII.—A. S., peddler, aged twenty-nine years. Drinks ten cups of tea daily. History otherwise negative. A year and a half ago, without apparent cause, he suffered from ptyalism, which frequently disturbed his sleep. There was no cough, no bronchial symptoms, no pharyngitis. After several months the above symptoms were partly replaced by choking sensations, usually present in the morning, and by frequent explosive belching attacks particularly after breakfast. He was often awakened from sleep by sensations of hunger and by intestinal rumbling. Subsequently he complained of palpitation and of "uneasiness" in the chest. Examination of the heart showed it to be of normal size; heart's action is rapid, regular, a soft systolic murmur accompanies the first sound. Examination was otherwise entirely negative.

This patient shows the vago-excitor phenomena in the ptyalism, the pharyngeal spasms, belching, and hungry sensation (gastric irritability). The rapid heart action and resultant slight cardiac dilatation and systolic murmur, I attribute to a reflex disturbance of the sympathetic, thus allowing unrestrained action of the cardiac accelerators.

The tea drinking is the only factor in the patient's history which points to an etiology for the vagus excitation.

It will have been noted that some of the statements I have made are based on purely theoretical grounds. This is unfortunately necessary since we are not as yet in possession of sufficient facts in connection with functional affections of the vagus or indeed of other nerves. By careful analysis of the less obvious symptoms of a series of cases I have attempted to correlate, to group together under a single condition many symptoms that have heretofore been unclassified, and to show the inter-dependence of apparently disjointed phenomena.

As also stated by Eppinger and Hess, it is possible that cases showing a marked susceptibility to vagal influences follow a familial type. Three of the cases here reported were two sisters and a son of one of them; two cases were mother and daughter. My observations, however, do not comprise sufficient cases to warrant any definite deductions.

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In conclusion, I would state:

1. The study of reflex phenomena is of great importance with reference to vagus excitation.

2. In some instances of gastric affections and of heart irregularities, a careful survey of the clinical picture and a search for apparently minor symptoms will reveal the significance of vagus excitation in the symptom-complex.

3. The discovery of such a symptom-complex is of special importance in prognosis and therapeusis.

4. Gastric ulcers may occasionally originate from functional vagus excitation.

5. Some apparently hysterical symptoms will be found to be functional (vago-excitive) and not neurotic in the ordinary sense of the word.

REVIEWS

AMERICAN PRACTICE OF SURGERY. EDITORS, JOSEPH D. BRYANT, M.D., LL.D., and ALBERT H. BUCK, M.D., of New York City. In eight volumes. Vol. VIII. Pp. 1146, with 406 illustrations. New York: William Wood & Co., 1911.

THIS is the last of a series of eight ponderous volumes comprising, in the words of the title page, "a complete system of the science and art of surgery, by representative surgeons of the United States and Canada." The first volume was reviewed in these columns only five years ago; and when the magnitude of the undertaking is considered, it is truly remarkable that it has been possible to complete the series in so comparatively short a time.

The present volume concludes regional surgery, comprising articles on intrathoracic surgery, by Joseph Ransohoff and J. Louis Ransohoff, of Cincinnati; surgery of the spleen, by Alex. E. Garrow, of Montreal; surgery of the kidneys and ureters, by James Bell, of Montreal; surgery of the pancreas, liver, gall-bladder and bile ducts, by Geo. D. Stewart, of New York City; that of the urinary bladder and prostate, by Alex. Hugh Ferguson, of Chicago; of the ovaries and Fallopian tubes, by Benjamin R. Schenck, of Detroit; of the uterus and its ligaments, by John B. Murphy and Frank W. Lynch, of Chicago; extra-uterine pregnancy, Cæsarean section, etc., by Lewis S. McMurtry, of Louisville; together with disquisitions on surgery and the law, by Stephen Smith of New York, and Sidney Smith, of the New York bar; on hospitals and hospital management, by Christian R. Holmes, of Cincinnati; military surgery, by Major Charles Lynch, United States Army; naval surgery, by Surgeon-General Charles F. Stokes, United States Navy; administrative railroad surgery, by James Alexander Hutchison, of Montreal; together with a brief appendix on the relation of blood pressure to surgery, by J. E. Sweet, of Philadelphia. There is also a general index to the entire eight volumes. This appears to have been constructed by a mechanic rather than a surgeon, and combines all the faults of the indices to the preceding volumes.

The article on intrathoracic surgery, as explained by the editors in a footnote, was not completed in sufficient time to be included in its appropriate place in Vol. VII; but it appears, nevertheless, to

have left the authors' hands too soon for them to make any mention of the method of anesthesia by "intratracheal insufflation," which bids fair to supplant the methods of "differential pressure." As this is the most notable advance in intrathoracic surgery during the past decade, it is a pity that it was overlooked. Brought prominently before the profession by Meltzer and Auer in 1909, it may be said to have become well recognized by the end of the year 1910. The article includes, besides surgery of the lung and mediastina (exclusive of the heart), some mention also of intrathoracic (aortic and innominate) aneurysms. This is well, as the monograph by LeConte and Stewart, in Vol. VII, does not discuss the individual forms of aneurysm; indeed, nowhere in the entire system can there be found any account of subclavian, popliteal, or other of the more important varieties. Carotid aneurysm is dismissed in a few lines in the article on the surgery of the neck, in Vol. VI.

In the otherwise admirable discussion of the surgery of the kidney and ureters, by Bell, one likewise looks in vain for any mention of acute septic infarct of the kidney, made so familiar a subject by the teaching of Brewer, of New York. Bell thinks renal tuberculosis usually primary in the kidney, and he advises nephrectomy when the patient is seen in the operable stage.

The contributions of Geo. D. Stewart, on the surgery of the pancreas, liver, and bile passages, call for little comment. They are sensible; and they form a safe guide for a surgeon who wishes to be neither hysterically radical nor hopelessly wedded to conservatism. On the subject of the pathogenesis, however, his views are not in accord with current teaching; he holds that most infections of the bile passages ascend the ducts from the intestine; he dismisses infection by way of the portal blood stream with a mere mention, and thinks the systemic circulation a very rare channel; and he successfully evades a discussion of the pathogenesis of pancreatic infections.

The late Alexander Hugh Ferguson lived to complete a characteristically positive and personal article of one hunderd and twenty pages on the bladder and prostate. He has made much use of Deaver's monograph, both in text and illustrations, but perhaps with too little credit. He condemns all operations for prostatic carcinoma, except the establishment of a suprapubic fistula by Hunter McGuire's method. While he prefers the perineal route for prostatectomy, he generously quotes, *verbatim*, at a length of over seven pages Mr. Freyer's own account of his method of suprapubic prostatectomy; and damns the Johns Hopkins operation, not so much with faint praise (for praise is absent from his lips), but by the assertion that the "conservative" feature of it "does not appeal to decent old men."

The subject of abdominal gynecology covers 320 pages, of which

by far the largest part (245 pages), written by John B. Murphy and Frank W. Lynch, is devoted to the surgery of the uterus and its ligaments. This is a very thorough article, but not so comprehensive as could be wished. Especially valuable is the discussion of uterine carcinoma; the authors approve the modified Wertheim operation, in "operable cases," and express their conviction of the value of the cautery and of chloride of zinc applications in those which are inoperable, which unfortunately in this country still form by far the larger class.

The monograph on surgery and the law is composed chiefly of court decisions, systematically arranged according to the legal questions involved, and should prove of great value to both lawyer and surgeon in preparation of medicolegal suits, expert testimony, etc.

Dr. Holmes, who is a member of the Building Commission of the New General Hospital in Cincinnati, has done the profession a distinct service in collecting so much valuable information as to hospital construction and management. He prefers the pavilion plan and urges that urban hospitals be established in suburban districts, or, at any rate, where there shall be plenty of air and light, with freedom from dust and noise; arguing that automobile ambulances, electric street cars, and other means of transportation render suburban points readily accessible for both patients and their friends. In this scheme, however, he fails to consider the requirements of hospital out-patients and accident cases, unless separate receiving stations are to be maintained in the thickly populated centres. No doubt the total abolishment of dispensaries would simultaneously abolish the "dispensary evil," but certainly it would work great hardship to the deserving poor, as well as to that large middle class, who, while they can afford to pay small fees, prefer to be reckoned among the paupers rather than receive the inefficient treatment which too often is all they can obtain outside of a hospital.

Reviewing these eight large volumes as a whole, it must be confessed that they scarcely fulfil the expectations of the subscribers. They are clumsy, both physically and intellectually. The paper is thick and brittle; the type is large, well leaded, and much unnecessary display typing is employed. By proper condensation of the merely physical attributes of the volumes their unwieldy size could be very materially reduced. The editing leaves much to be desired. To ascertain the author of any article in the entire system one must turn to the initial page of the article in question; then to ascertain who he is (for, unfortunately, many of the authors are not as truly "representative" as the title page suggests) one must turn again to the list of authors at the beginning of each volume. It surely would have been much better, and certainly very easy, to include in the table of contents both the

authors' names and their "credentials." It was quite needless to repeat the legend "American Practice of Surgery" at the top of each of the 7152 left-hand folios which the system contains; the subject of the special monograph should be stated here, and its subheadings on the right hand pages; there is no one who does not know how much more useful a volume is when thus arranged. These are clear faults of editing. Since the publication of the last volume the publishers have forwarded to the subscribers small slips of subject headings to be pasted on the backs of the volumes, thus adding greatly to the ease with which a given subject may be found; but there seems no reason why these headings should not have been printed on the backs of each volume as it issued from the press. For four years it was necessary, before finding the subject desired, to consult the index of each of the volumes published up to date.

The individual articles (one hundred and two in number) contained in these eight volumes are, as might well have been expected, of very varying importance and value. Some of the authors (fortunately not a majority) seem to have had no other object in view than to get through with their task with the least possible expenditure of energy and intellectual acumen; they have scribbled down a lot of ideas worthless alike from their lack of logical sequence and from their innate inanity; while others have gone at their task seriously and conscientiously, and have furnished in some instances brilliant, in all cases worthy, contributions to the science of surgery.

It is well at the conclusion of every endeavor, literary or other, to bear in mind two precepts: the first, that no matter how well the assigned task has been performed, the workers have done nothing but their duty, and that they are therefore "unprofitable servants;" the other, that well-known quotation from Seneca the Philosopher: *Multum restat adhuc operis; multumque restabit, nec ulli nato post mille sæcula præcludetur occasio aliquid adjiciendi.*

A. P. C. A.

THE LIFE HISTORY, FUNCTION AND INFLAMMATION OF THE APPENDIX. By EDRED M. CORNER, M.A., M.C., F.R.C.S., Surgeon to the Surgical Isolation Wards and Out-Patients, St. Thomas's Hospital Pp. 23. London: John Bale, Sons & Danielsson, Ltd., 1911.

THIS little volume is a reprint of an address delivered at the Clinical Society at Manchester, January, 1911. This address might have been interesting to a group of laymen but contains nothing new that would interest physicians. The first portion of the text is devoted to a discussion of the relation of bread, made

from flour rolled by big steam rollers of a fluted type, to the etiology of appendicitis. Corner does not, of course, believe that appendicitis is solely due to our daily bread, but does believe that since the introduction into England of American methods of preparing flour, appendicitis in that country is on the increase. One might remark that there are other reasons. The remainder of the booklet is devoted to a discussion of certain well-known facts, namely, the advisability of removing the appendix if diseased; the existence of "unconscious, undiagnosable, unrecognizable appendicitis," which perhaps is only our old friend appendicitis larvata; the gradual atrophy in most people after the age of twenty years of the lymphoid tissue of the appendix, a fact pointed out by Ribbert many, many years ago; finally, the increasing frequency with which carcinoma of the appendix is detected histologically.

Perhaps the contents of this book seem trite to an American for the reason that "in America it is very common, indeed, to have the appendix removed on a comparatively slight provocation." But, as a result of this rashness, we have learned long ago the facts which Mr. Corner now teaches. G. P. M.

FOOD AND THE PRINCIPLES OF DIETETICS. By ROBERT HUTCHISON, M.D. (Edin.), F.R.C.P., Physician to the London Hospital, etc. Third Edition; pp. 615; 3 colored plates; 33 illustrations. New York: William Wood & Co., 1911.

A SERIES of lectures addressed to the students of the London Hospital formed the nucleus for the first edition of this work. The reception given these lectures and the almost total neglect of the subject of dietetics in ordinary medical education induced the author to publish them in book form. In recasting them for publication, however, a large amount of additional matter has been used. In the present edition the whole book has been thoroughly revised and the chapters dealing with the "Use of Diet in Disease" considerably enlarged. A new chapter has been added on certain "Dietetic Cures and Systems."

The work is thoroughly scientific and up to date, and the conclusions are based on the results of scientific research so far as they have been worked out at the present time. Much still remains unsolved in our ideas of the assimilation of food by the body in health and disease. At the present time of close inspection of proprietary foods the chapter devoted to "Artificial and Predigested Foods" is of particular interest, for the author has attempted to arrive at an unprejudiced estimation of their value.

The previous editions of the work have already held a prominent position among books of reference on this subject, and the present

volume promises to retain this place. The book is very readable and is of unusual interest in the way of general information about foods, even to lay readers. The typographical errors are few, the printing clear and distinct, and the index unusually full and complete, making reference to any of the numerous subjects easy. We can well recommend the book as an excellent work on the subjects of food and dietetics, and as such it will appeal not only to students and instructors in these special subjects, but should be of great help to physicians and students of medicine who wish to obtain the full benefit of food in the preservation of health or in the treatment of disease. F. H. K.

ORTHOPEDIC SURGERY. By EDWARD H. BRADFORD, M.D., and ROBERT W. LOVETT, M.D. Pp. 410; 364 illustrations. New York: William Wood & Co., 1911.

THE present volume is much smaller than those issued by the same authors some years ago. It is an entirely new book. They present it as "a condensed handbook for the use of students and practitioners, embodying a brief statement of the generally accepted opinions as to the nature and treatment of the affections under consideration."

Viewed from their standpoint the book is certainly satisfactory. The subject is presented in a systematic, careful, and clear manner that cannot fail to be suitable for those who are to a great extent ignorant of this branch of surgery.

It seems to be most admirably suited for a students' text-book. Its conciseness, its brevity, and the clear, lucid descriptions and abundant illustrations all recommend it to that class. Students have not the time to study large treatises, or to go into the subject exhaustively. The time has passed when the subject of orthopedics can be adequately treated in works of general surgery. It is imperative that the student possess some such book as this and familiarize himself with its contents. When, however, the student enters upon the practice of medicine, some of the features that commend the book to the undergraduate tend to make it less satisfactory for the practitioner. The short, brief, concise, and didactic descriptions and directions are not sufficient for the physician, who wishes to thoroughly inform himself about an individual affection or class of cases. He should supplement it with a larger work, in which sufficient space is given to detailed explanations and directions.

The views expressed throughout the book are preëminently those of the Boston school of orthopedists, and though sound, are naturally conservative. To have presented more fully the operative or

more radical side of orthopedic surgery which is developing so rapidly would have been impossible without unduly enlarging the size of the book, and thereby impairing its value as a text-book.

It is stated that while synovial tuberculosis exists in a large majority of the cases the affection arises in the bone. In view of the deaths that have been caused by applying pure carbolic acid to the tissues, we think it would have been wiser to have omitted mention of its use in the operative treatment of hip disease. We doubt the advisability of Bardenheuer's excision of the acetabulum, even as a life-saving measure. We think it would be well to mention multiple cancellous exostosis, probably more common than myositis ossificans. The failure to recommend cod liver oil in rickets should be remedied. We fail to find any mention of the reviewer's method of reducing congenital luxations of the hip by placing the child on its face and pushing the head of the femur into place; thus is our vanity hurt. Nothing is said about the use of outside braces for club feet, perhaps this was intentional, but if so we must dissent. On page 355, fifth line from the bottom, the word "tendon" is used instead of "knife," and on page 203 "Diagnoses" occurs twice in large type.

In conclusion we commend the book thoroughly to the profession, and suggest that every general surgeon secure a copy and read it carefully, as thereby he will acquire a faint idea of what ought to be done for the too many cripples that are still in need of proper treatment. G. G. D.

PUERPERAL INFECTION. By ARNOLD W. W. LEA, M.D., B.S., F.R.C.S., Lecturer in Obstetrics and Gynecology, the University, Manchester; Surgeon to the Northern Hospital for Women, Manchester. Pp. 384; 23 illustrations and 36 plates. London: Oxford University Press.

IT is impossible to do justice to this work without seeming to flatter. No adverse criticism has any place. It is complete in grasp and definite in teaching, and, best of all, it is eminently sane. We believe that the above will be the verdict of those men whose experience has made them competent to form a judgment.

The author opens the work with a most interesting historical review, and then devotes a chapter to the classification of the various forms of sepsis. In accord with present-day teaching he classifies as toxemias all septic processes uncomplicated by deep penetration or blood invasion of organisms, whether or not said organisms are pathogenic or putrefactive.

The third chapter is concerned with the frequency, mortality, and morbidity of sepsis, and if there were nothing else of value to

commend the book, the compilation of this statistical chapter would make it worth while. Unfortunately the general mass of the profession, in whose hands lies the bulk of obstetrics, are prone to belittle both the bitter experience of the past and the scientific testimony of today, and, continuing to worship before the tablet of mercury in a dirty basin of unboiled water, still needlessly jeopardize human lives. This chapter should be read by every man engaged in this branch of medical work. In drawing his conclusions to this chapter the author gives three rules by which puerperal morbidity in obstetrics may be greatly reduced, namely, by the complete disinfection of those attending cases of labor, by efficient disinfection of the external genitalia during labor; by limiting the examinations so far as possible, and by the avoidance of all unnecessary operative interference. It would be well if the second of these rules could be branded upon the conscience of every one engaged in midwifery practice. Consulting experience teaches that there is a passable observance of the first and last of the three rules mentioned above, but that the majority of even intelligent men have no conception of adequate external cleansing, and, therefore, naturally cannot practice it.

The chapters upon bacteriology and etiology are up to date, and will well repay careful reading. Among other interesting questions discussed, perhaps the most interesting is whether pathogenic organisms may be found in the uterine cavity of normal cases at various intervals after delivery, and also that while autogenetic infection is rare, as a cause of severe sepsis, its occurrence probably explains a number of cases of slight pyrexia in the early puerperium. This whole question is of the greatest interest, but as yet cannot be looked upon as at all definitely settled.

The chapters upon general pathology, pathological anatomy, and the symptoms of sepsis are the result of painstaking care in preparation. It gives particular pleasure to note that the relation between the polymorphonuclear and eosinophile cells is insisted upon as a prognostic sign of value in severe cases.

A separate chapter is devoted to localized infection of the generative tract, and in the next succeeding chapter the prophylaxis of infection is carefully elaborated under three subdivisions, namely, during pregnancy, during labor, and during the puerperium. While there is nothing new in this chapter there is a great deal which should be assimilated and practised, particularly by those men to whom obstetrics is but a small part of their daily work. In speaking of the use of gloves, however, there is one point which cannot be passed without adverse comment, namely, the statement that their use makes it difficult to detach membranes or portions of placenta from the uterine wall. To this the reviewer cannot subscribe, since a little perseverance will enable any one to become as expert with the gloved as with the bare hand. The increased

safety offered by their use in intra-uterine manipulations, which by the way is definitely stated on the succeeding page, should have excluded this objection.

In his consideration of the treatment of infected cases the author lays stress upon the fact that there is no specific, and calls attention to the results obtained by purely expectant handling. Isolation, rest, nutrition, and the use of rubber gloves are insisted upon as general principles.

It is interesting to note that écouvillonage, or the use of the intra-uterine brush, is recommended as a valuable means of cleansing the uterine cavity. Good results from this method are reported by the author, and he claims avoidance of certain dangers inherent in other instruments. He gives a well-considered section on the use of the curette, with careful enumeration of its possibilities for harm, and while here, as elsewhere throughout the book, the judicial, or rather non-partisan attitude, is maintained, it is plain that his opinion is adverse to the curette, in acute cases at least. This is in accord with the view of most competent men of the present day. It is somewhat surprising that the valuable curettement forceps of Emmet is not mentioned, since this instrument is free from the objections rightly urged against the curette, and, theoretically at least, seems preferable to the écouvillon.

Readers of this chapter will note with interest that in the treatment of localized pelvic inflammation without suppuration, the Bier method is advocated. The general treatment of infected patients receives adequate attention, and it is interesting to note the unfavorable criticism of alcohol. What a shock the present-day attitude regarding this drug would be to the active men of a generation ago!

A full consideration of the various means to produce leukocytosis, as nuclein, saline solution, and colargol, is given, but, of course, no definite statements are permissible as to their specific value. After a very clear and concise enunciation concerning the much vaunted antistreptococcic sera, the conclusion is reached that their value is at best questionable. The hope is, however, expressed that there will be developments along this line, with better results in the future.

In the chapters upon the operative treatment there is nothing new, but the conservatism is praiseworthy, and we are particularly glad to note this quality in the advice given regarding acute pus tubes and ovarian abscess, and also in the narrow indications admitted for hysterectomy. We can not close this review without again expressing our admiration for the work. It is by far the best book upon this subject as yet published, and should have a wide circulation. W. R. N.

PRACTICAL PATHOLOGY. By ALDRED SCOTT WARTHIN, Ph.D., M.D., Professor of Pathology and Director of the Pathological Laboratories in the University of Michigan. Second edition; pp. 321; 55 illustrations. Ann Arbor: George Wahr, 1911.

THIS is a manual of autopsy technique written primarily for students, but valuable to anyone engaged in such work. Part I, comprising 198 pages, is a very thorough consideration of the autopsy proper. Part II, comprising 111 pages, describes the laboratory treatment and examination of material obtained at autopsy. The preliminary portion of Part I, containing the legal restrictions relating to autopsies, the order of autopsy and a list of structures that should be examined, suggestions as to protocols, and the external examination and the signs of death, is unusually complete. The description of removal and examination of the organs is clear and thorough. For the removal of the spinal cord both posterior and anterior methods are described, and for opening the heart four methods are given. After the removal of each viscus, the pathological changes especially to be looked for in the organ are noted. A chapter is devoted to the autopsy of the newborn and another to the medicolegal autopsy, with a description of the characteristic lesions due to twenty-five common poisons.

In Part II the use of the freezing microtome and all of the common methods for fixing tissues are described, with the especial advantages and disadvantages of each. At least one staining method, and, as a rule, several, is given for each kind of tissue, and for each kind of degeneration, infiltration, and exudation; no attempt is made, however, to give all the modifications of a staining method. Bacteriological methods are not considered except the staining of microörganisms in tissues and in smears, with the staining reaction of about forty common bacteria, yeasts, and animal parasites.

About half the illustrations are of autopsy instruments and common laboratory apparatus; however, the remaining ones are well selected to illustrate methods of exposing, or examining, various organs. The material in the book is clearly presented, well arranged, and adequately indexed. J. H. A.

REVUE DE MÉDECINE MÉMOIRES RÉDIGÉS EN L'HONNEUR DU PROFESSEUR RAPHAËL LEPINE A L'OCCASION DE SA RETRATE PAR SES ELÈVES ET SES AMIS. Pp. 885; 20 illustrations. Paris: Félex Alcan, 1911.

OPPOSITE a full-page portrait of Professor Lepine is found this dedication with which the book opens: "To the Master who being a scientist wished to be always a physician, to him who

has been the respectful servant of observation, but who has taken Pathological research as a foundation for all and who has taken as a starting point for all medical action Physiology, Pathological Anatomy, Biological Chemistry and Experimentation, to him who has taken a great part in the revival of Medicine and who still preserves it in his studious retirement, to the educator who has said, 'The physician should think physiologically,' to Professor Raphaël Lepine."

It is in these words that his pupils, associates, and admirers dedicate this volume in honor of Professor Lepine on the occasion of his retirement after over thirty years' continuous service as a member of the Medical Faculty of Lyons. It was about 1875 that Lepine, who as one of Charcot's brilliant pupils was well fitted for the position, was called to a chair of the Medical Clinic at Lyons, directly concerned with the introduction of scientific investigation and laboratory methods. His success was remarkable, as a glance at the breadth of scope and progressiveness of his bibliography would suggest.

The collection of papers gathered in this volume consists of one hundred and twelve contributions, the majority by members of the Faculty of Lyons, but including also prominent writers from other parts of France, Italy, and England. The subjects are as various as the interests of the many authors, and no attempt has been made to confine the articles in any way so that many concern the specialities. As a whole, the volume commands respect as must any group of papers which contain in the list of its authors such names as Achard, Lauder Brunton, Favre, Patella, Roger, and Weill.

To attempt to select individual articles for commendation from such a number is difficult and reflects merely the reviewer's personal interests. Such is the contribution of Maurice Letulle, Professor to the Faculty of Medicine of Paris, on Cysts of the Kidney. In this 25-page monograph is a clear detailed description of the various types of cyst found in healthy and pathological kidneys with illustrations of microscopic sections through them. The great number of the articles and the great diversity of subjects, if nothing else, make this book of great interest, for although no epoch-making contribution is contained and but little original work reported, yet we may here make the acquaintance of a group of the French medical teachers and writers and obtain a sort of bird's-eye view of French medical thought.

The volume is very creditable and a fitting tribute to a man who not in his own country alone, but throughout the entire scientific world is held in high esteem and honor. O. H. P. P.

THE ORIGIN OF LIFE. By H. CHARLTON BASTIAN, M.D., F.R.C.S., Emeritus Professor of the Principles and Practice of Medicine. Pp. 119; 10 plates. London: University College, 1911.

THE author presents in this book some researches which the British Royal Society found unsuitable for acceptance. He has demonstrated the appearance of a few torulæ, bacteria, and moulds in hermetically sealed tubes, containing a solution of sodium silicate with either ammonium phosphate and phosphoric acid, or liquor fevri nitratis, in from four weeks to twelve months after the sealed tubes have been heated in a bath of from 100° to 130° C. for ten to twenty minutes. He has further shown that these organisms so obtained are alive, by transplation into sterile bouillon with immediate abundant growth of the organisms in this medium. A careful reading of the book, however, fails to show that the author has determined the temperature attained within the sealed tubes during the so-called sterilization, nor does he report any control experiments to rule out the possibility of having merely lowered, without extinguishing, the vitality of organisms originally present in the tubes. The work in consequence would seem to be, until further controlled, of but little value for the drawing of any definite conclusions and certainly does not warrant the author's assumption that his findings are an evidence of spontaneous generation. The title of the book is, therefore, misleading. The plates are photomicrographs of some of the organisms found in the tubes. J. H. A.

MINOR SURGERY. By L. A. BIDWELL, F.R.C.S., Surgeon of the West London Hospital; Dean of the Post-graduate College; Consulting Surgeon to the Blackheath and the Charlton Hospitals. Pp. 205; 88 illustrations. London: Hodder and Stoughton and Frowde, 1911.

THE work being a manual aims to give simple, clear, practical directions for the performance of every-day minor surgical work. No attempt has been made to give the several methods that may be useful in accomplishing the desired result, but rather the author has given that which he, in his experience, has found to be the best.

Emergency operations of the minor and a few of the major type are described in practical detail. In many instances the author has given interesting facts not generally known or appreciated.

The work is a valuable one for hospital internes, students, and the busy general practitioner, as it contains many facts that can be obtained only through association with some one doing a large amount of this work. Furthermore, it describes many of the

minor practices which the larger works deem too elimentary for consideration.

The book is well written and the style is pleasant. The facts are condensed and there is no useless reading of theories, possibilities, or diverse opinions. E. L. E.

AN INTRODUCTION TO SURGERY. By RUTHERFORD MORISON, M.A., M.B., F.R.C.S., Professor of Surgery, University of Durham. Pp. 162; 146 illustrations in the text and 5 colored plates. New York: William Wood & Co., 1911.

THIS book is an abstract of the author's lectures on surgery and is an interesting summary of the modern point of view of the subject discussed. It includes inflammation, ulcers, gangrene, syphilis, tubercle, malignant disease, hemophilia, wounds, certain disturbances of the abdominal and pelvic viscera, and certain indications for operation. The general scheme or outline of the book is most unsatisfactory, but the subject matter contained therein is important and discussed in a most interesting manner. The work should be exceedingly valuable to those who are students of Mr. Morison and contains a number of chapters of which use may be made with advantage by teachers of surgery, but to the general practitioner or to the student at large it is apt to prove unsatisfactory, owing to the incompleteness of the subject discussed and to the failure to include many important things. The illustrations are excellent and the general make-up of the book very satisfactory.

G. P. M.

THE MECHANISM OF LIFE. By Dr. STÉPHANE LEDUC, Professor a l'école de Médicine de Nantes. Translated by W. DEANE BUTCHER, Formerly President of the Röntgen Society, and of the Electro-therapeutical section of the Royal Society of Medicine. Pp. 172; 63 illustrations. New York: Rebman Company, 1911.

THIS interesting book gives a clear presentation of some of the more important physical and physicochemical laws that must be taken into account in considering organic life and its phenomena, including the laws of colloids. It also presents some relatively new observations on diffusion and on osmotic growths that may become an aid in the interpretation of phases of cellular activity. These osmotic growths are inorganic products produced under certain experimental conditions which, in structure and activities, show an extraordinary analogy to simpler forms of organic life.

Some kinds have been produced by many observers, and the author gives directions for repeating such experiments as are original with him. At times the author draws from his observations conclusions which his readers will probably not accept. This is especially true of his speculations upon the nature of light, upon spontaneous generation, and upon the cause of the rhythmic character of the heart beat. He is also too ready to explain by simple physicochemical laws alone, some of the most complex of cellular activities. One cannot read the chapter on karyokinesis, however, without feeling that the observations presented must be of great significance in explaining this phenomenon. The illustrations are photographs of the forms obtained in diffusion experiments and of osmotic growths and are most interesting.

As the result of collaboration between the author and the translator this English edition has some advantage over the original French edition.

The book will well repay a careful reading. J. H. A.

THE PROBLEM OF RACE BETTERMENT. By J. EWING MEARS, M.D., LL.D. Pp. 45. Philadelphia: Wm. J. Dornan.

WITHIN this title are included three papers, published in various medical journals, and the subject matter contains only about 40 pages. The subject, however, is extremely well handled and forcibly written. It is needless to say that everyone will agree with the author that something should be done to diminish the ever-increasing number of idiots, imbeciles, criminals, and epileptics who are constantly born into what seems to be a lawless sphere, at least in so far as this particular subject is concerned. However, since the publication of the first of these papers, laws have been enacted in some of the States to desexualize this class of patients, notably in the State of Indiana. Because of the very nature of our Republic in so far as matters of health are concerned, we are woefully back of the more central types of government, such as Germany, which readily control social questions. It is simply another argument for the establishment of a national Bureau of Health and of a Secretary of Health in the Cabinet of the President. T. H. W.

LES SYNCINESIES. By LE DOCTEUR G. STROEHLIN, Ancien Interne des Hôpitaux de Paris. Pp. 147. Paris: G. Steinheil, 1911.

THIS is a thesis upon the general subject of associated movements. No new conclusions are presented. T. H. W.

PROGRESS

OF

MEDICAL SCIENCE

MEDICINE

UNDER THE CHARGE OF

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The Effects of Subdural Injections of Leukocytes on the Development and Course of Experimental Tuberculous Meningitis.—MANWARING (*Jour. Exper. Med.*, 1912, xv, 1) finds that, though the mechanism of cure for tuberculosis has not been fully worked out, it would appear that the phagocytic cells play an important part, since it has been shown that leukocytes injected simultaneously with tubercle bacilli have the power of decreasing the pathogenic properties of these organisms. Manwaring has conducted experiments on dogs to study the effects of injected leukocytes on the development and course of experimental tuberculous meningitis. The cerebrospinal membranes are almost impervious to the blood proteids, and so normally receive little or no protective substance from this source, and few, if any, leukocytes. Hence the meningeal cavity is always in an inferior condition of defence against infection. His observations consisted in trephining dogs, and after healing, injecting them intradurally with strains of varying virulence of bovine and human tuberculosis. At the same time in certain animals, sterile leukocytes, obtained by the intrapleural injection of turpentine, were injected. By the injection of tubercle bacilli in this way, a disease is produced after five to thirty days' incubation, characterized by increasing paralysis, and terminating almost invariably in death. The pathological picture varies with the virulence and dosage of the organisms. It shows meningeal thickening with adhesions. The subdural space usually has an active tuberculous process varying from an accumulation of fluid, fibrin, and semi-necrotic cells, to well-organized masses of diffuse tuberculous tissue. Tubercle bacilli are present. Manwaring found that if leukocytes were

injected with organisms, though one-quarter of the animals died within a few hours—many with no accessory cause found—in those that lived there occurred uniformly a marked delay in the development of a paralysis, and a marked prolongation of life depending on the virulence of the organism used.

Intramuscular Injections of Defibrinated Blood in Anemia.—P. Esch (*Deut. med. Woch.*, 1911, xxxvii, 1943) reports the use of intramuscular injections of defibrinated blood in the treatment of anemia. Human blood is employed, and the injections are made into the gluteal region. As much as 30 c.c. are given at a time. It is important, Esch says, to determine that the end of the needle is not in a vein, so that intravenous injection may be avoided. Favorable results are reported in a case of pernicious anemia following pregnancy and in secondary anemias.

The Incoagulable Nitrogen of the Blood in Nephritis and Uremia.—H. Hohlweg (*Deut. Arch. f. klin. Med.*, 1911, civ, 216) has studied the incoagulable nitrogen (*Reststickstoff*) of the blood serum in nephritis, using the method devised by himself and H. Meyer. His results confirm those of Strauss and several other workers. The blood was always obtained between 10 and 11 A.M., the patient receiving only a cup of milk and a roll for breakfast, so that the effect of digestion might be fairly constant. About 150 c.c. of blood were withdrawn from an arm vein and placed in the ice-chest to allow the serum to separate. The serum was then used for the determinations. It was found that the normal average incoagulable nitrogen is 0.051 gram per cent., the extremes being 0.041 gram and 0.060 gram. The results of the study summarized as follows: (1) The incoagulable nitrogen of the blood serum of patients without renal diseases averages 0.051 gram per 100 c.c. serum; of this 11.7 per cent. is precipitable by tannin; 27.4 per cent. is not precipitable by tannin; 60.8 per cent. is urea. (2) In nephritis the incoagulable nitrogen rises to 0.63 to 0.93 gram per cent. Essential differences in the values in parenchymatous and interstitial nephritides are not discoverable, either in the total incoagulable nitrogen or in its fractions. (3) The values found in nephritis exhibit no noteworthy increase when uremia develops, so long as the patient's condition is susceptible of improvement. Thus, total incoagulable nitrogen of 0.060 to 0.095 gram per 100 c.c. makes the prognosis relatively good, provided other complications, such as cardiac disease do not appear. (4) In the last weeks or months of life of a nephritic, the incoagulable nitrogen of the serum increases rapidly or slowly to the highest values known, whether or not uremic symptoms exist. The increase is progressive to the time of exitus. (5) These enormous collections of incoagulable nitrogen—up to 0.370 gram per cent.—are due almost wholly to increase of the urea fraction, which may amount to 80 per cent. of the total incoagulable nitrogen. There is a slight increase in the fraction which is not precipitable by tannin. No change is observed in the tannin-precipitable fraction. (6) The increase of the incoagulable nitrogen is merely the expression of renal insufficiency, and is in no way specific for uremia. (7) In nephritics who die of other diseases, as affections of the heart, no such striking accumula-

tion of the incoagulable nitrogen is seen in the serum. In patients without renal disease, normal values are found, even in the last hours of life. Increase of the incoagulable nitrogen above 0.120 grams per cent. is characteristic of nephritis which will soon terminate fatally. (8) In cases of doubtful diagnosis, the quantity of the incoagulable nitrogen may be of aid. In prognosis, too, valuable information may be gained.

The Relation of Bovine Tubercle Bacilli to Human Infections.—H. KOSSEL (*Deut. med. Woch.*, 1911, xxxviii, 1972) contributes a study of the relative frequency of infection of man with human and bovine tubercle bacilli. Of 709 patients with pulmonary tuberculosis, two cases were definitely bovine alone, and probably a third, in one case both human and bovine bacilli were demonstrated, while in 705 instances the human type of bacilli alone could be found. Such findings as these indicate beyond question the correctness of Koch's views that human tuberculosis owes its widespread prevalence to direct transmission from man to man. The elimination of the bovine source would cause a scarcely appreciable reduction in the incidence of the disease.

Acetonuria in Diphtheria and Acute Anginas.—F. REICHE (*Münch. med. Woch.*, 1911, lviii, 2153) has contributed valuable statistics on the occurrence of acetonuria in diphtheria and acute anginas. In all, the urine from 3826 patients has been studied. Of these, 3200 were acute diphtheria with Löffler's bacilli in the exudate. Acetonuria was demonstrable with the sodium nitroprusside test applied directly to the urine in 2079, or 65 per cent. of the cases. The duration of the acetonuria was usually only a few days, at times one day; less often it persisted a week or more. The great majority of all cases of acetonuria occurred between the first and fourth days of the disease. Since many patients entered the hospital late in the disease, it is quite possible acetonuria had existed earlier, but had disappeared before urinary examinations were begun. Most of the cases were found in patients under fifteen years of age. The height of the temperature has little effect, but acetone is much commoner in the severe forms of the disease. Diet seemed to play a small part in the production of acetonuria, since many of the patients exhibiting it were receiving carbohydrates. Among 390 cases of follicular angina, 159, or 40.8 per cent., showed acetone in the urine. In 172 cases of angina simplex, acetonuria occurred in 44, or 25.6 per cent. Sixty-four cases of quinsy were studied. In 25 the abscess had been incised or had ruptured previous to admission. In all of the remaining 39, acetonuria was present.

The Technique of Hemoglobin Determinations.—C. STAUBLI (*Münch. med. Woch.*, 1911, lviii, 2429) has noticed, in using the Autenrieth and Königsberger colorimeter for hemoglobin determination, that the results vary according to the time interval between diluting the blood with tenth-normal HCl and making the reading. As the blood-HCl mixture stands, its color darkens. This observation led to a thorough investigation of the phenomenon. In using the Autenrieth and Königsberger colorimeter, 20 c.mm. of blood are mixed with 2 c.c. of tenth-normal HCl. After preparing this mixture, Stäubli made colori-

metric determinations at the end of one-half, two and one-half, five, ten, fifteen, twenty, twenty-five, and thirty minutes. He found that the mixture becomes progressively darker. The darkening of the fluid is more rapid in the earlier minutes. By plotting the values found, the curve obtained is a parabola, and from a study of this Stäubli recommends that the reading be made at the end of ten minutes. After this time has elapsed the darkening of the solution is slight and the error small. Greater relative increases are observed when the hemoglobin is low than with high values. In using the Sahli hemometer, 20 c.mm. of blood are diluted with 0.2 c.c. of tenth-normal HCl (*i. e.*, 10 divisions of the graduated tube), and after one minute the contents are diluted with water till the color is like that in the standard tube. Here the strength of the HCl is so greatly diminished by dilution with water that darkening of the solution is much less pronounced than with the Autenrieth-Königsberger colorimeter. It is essential, however, that Sahli's directions be rigidly adhered to. (1) Tenth-normal HCl is filled into the graduated tube exactly to the mark 10. A greater amount of acid will accelerate the darkening. (2) The acid should be tenth-normal. (3) Wait exactly one minute after adding the blood, and then dilute with water. The dilution with water inhibits the darkening effect of the acid. (4) Stäubli would add a fourth direction, *i. e.*, to make a maximal dilution with water as quickly as possible. He suggests that a still better procedure for determining hemoglobin with Sahli's hemometer is the following: The blood-HCl mixture is diluted at once with tenth-normal HCl till the color is approximately that of the standard tube; then wait ten minutes to make the final colorimetric determination. This final dilution, which will require only a few drops, may be made either with water or acid. With this technique the results are uniform. Whatever method is followed, it is absolutely essential that it be adhered to strictly, in order that the results may be comparable.

The Influence of Some Foodstuffs on Gastric Secretion.—F. Craven Moore and H. E. Allanson (*Proc. Roy. Soc. Med.*, London, 1911, Medical section, 19). The observations of Pawlow and his co-workers having established the varying influence of different diebetic substances on the rate of gastric secretion, and the activity of the secreted juice in the dog, it was accepted as at least highly probable that a similar relation would obtain in the human, and in a limited fashion the truth of this has already been established. It seemed of some practical importance to determine by observation in humans, the effect on gastric secretion of certain foodstuffs which have been credited empirically with having some particular relation (ameliorative or causal) with certain disorders of the stomach. Fifteen individuals, including normal subjects and those with and without definite gastric disorder were used. A test breakfast of 4 breakfast biscuits, 10 ounces of tea, coffee, cocoa, without milk or sugar; milk, albumin water containing the white of one egg in 10 ounces of water, 2 teaspoonfuls of meat extract in 10 ounces of water, or 2 ounces of whisky in 8 ounces of water were given on a fasting stomach. The contents were removed an hour later, and tested for free HCl and total acidity. On the average, not less than three experiments were made with the particular substance under

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and in those for which the urogenital system and rectum are responsible, the treatment should be limited principally to the nervous system.

Hogge (*Ann. d. mal. d. org. gén. urin.*, 1911, ii, 2145) says that in most cases the phosphaturia is probably due to a secretory neurosis of the kidneys; at least one can say that the phenomena appear to be due to an influence upon the renal nervous system, which modifies the reaction of the urine. Aside from the diabetic phosphaturia of Teisser and the juvenile phosphaturias of the German writers, phosphaturia ought to be considered as a benign symptom from the point of view of the internist. In general one can say that phosphaturia is to be compared with azoturia, and that it is found in acute consumptives. In the chronic consumptive conditions, on the contrary, the organic combustion is more economical, and there is at the same time as hypoazoturia, a diminution in the elimination of the phosphates. From the surgical point of view, phosphaturia aggravates and complicates inflammatory diseases and lithiasis of the urinary apparatus. The best symptomatic treatment of phosphaturia consists in the administration of abundant liquids, of urotropin, in the choice of foods and in rules for hygiene and diet. Permanent phosphaturia indicates always an important disturbance in the general nutrition.

Alcohol Injection of the Gasserian Ganglion for Trigeminal Neuralgia.—Harris (*Lancet*, January 27, 1912, 218) says that during the last three and one-half years he has seen 90 cases of intractable trigeminal neuralgia, 86 of whom he has treated with alcohol injections. In only 3 cases was no relief obtained owing to the nerve not being found. In 3 others slight definite improvement resulted, and in the remaining 80 cases complete relief was obtained for intervals varying from as little as two months and four months in 2 cases, to two and one-half years and more. For the majority, twelve months of complete relief from pain may be looked for as a minimum, if the nerve is definitely hit and anesthesia produced; while two to three years and more may be expected when the depth of anesthesia indicates that the nerve has been properly destroyed. Strong alcohol injected into any nerve trunk destroys the nerve fibers almost immediately, and total loss of function will occur until regeneration takes place. The extirpation of the ganglion gives a permanent cure, but its severity deters a large number of sufferers from seeking relief by this means. Harris has tried to improve on the technique of the deep injection of of alcohol by carrying the alcohol through the foramen ovale into the Gasserian ganglion itself, in order that by destroying the nerve cells in the ganglion regeneration of nerve fibers along degenerated trunks shall be prevented, and a permanent cure be thus obtained. During the last fifteen months he has injected the ganglion through the foramen ovale with 90 per cent. alcohol in 7 cases. The first was done thirteen months ago, and he still remains perfectly well, with no pain of any kind. Harris' technique for injecting the Gasserian ganglion varies only in detail from that of injecting the inferior maxillary nerve at the foramen ovale; instead of inserting the needle close below the lower border of the zygoma, he chooses a point either on or slightly below the line joining the incisure notch to the ala nasi. This line in the average skull, when the teeth are in position, corresponds to the lower

border of the sigmoid notch of the lower jaw. To reach the foramen ovale, therefore, the needle must be directed slightly more upward, making it thus easier to pass the point through the foramen. As soon as the nerve is reached at the foramen, he injects it with 90 per cent. alcohol, using about 1.5 c.c., but if no general anesthesia is being given, he precedes the injection of alcohol by injecting 6 or 7 minims of 2 per cent. eucaine solution into the nerve, thus rendering the subsequent injection of the alcohol comparatively painless. If there results, immediately, deep anesthesia of the lower lip and chin from injecting the nerve itself, he then feels for the lip of the foramen ovale with the point of the needle, if this has not already been felt. Sometimes the needle slips easily through the foramen, sometimes it has to be worked through, but no degree of force should be used. Sometimes after pushing the needle through the foramen ovale, the point of the needle may leave the nerve tissue and enter the cave of Meckel, and cerebrospinal fluid may drip from the needle. If the injection of alcohol be then commenced no resistance will be felt to the push of the piston, and instantly the patient will complain of severe headache at the base of the skull and back of the head. Should this occcur the needle must be partially withdrawn, and then worked through at the posterior edge of the foramen, so as to keep the point within the substance of the ganglion. Injection for the second division at the foramen rotundum is usually more difficult than for the third division at the foramen ovale, and in a certain proportion of cases it is impossible. There is also more likelihood of hematoma from wounding the internal maxillary vessels. These points are additional reasons for preferring the injection of the Gasserian ganglion through the foramen ovale.

The First and Last Kink in Chronic Intestinal Stasis.—LANE (*Lancet*, December 2, 1911, 1540) calls particular attention to the last kink or obstruction in our drainage scheme which affects the large bowel where it crosses the brim of the pelvis on the left side. It is so arranged as to oppose the backward passage of feces from the pelvic colon into the large bowel, which is likely to take place if the kink or obstruction is not efficiently developed. When Lane first performed ileocolostomy for chronic intestinal stasis, he divided the ileum and put it into the sigmoid loop. At the same time he took great precautions to free the obstruction at the plevic brim referred to. This operation was frequently followed by backward passage of material along the descending, transverse, and ascending colon to the cecum, often necessitating the removal of the large bowel at a later date. This led to the removal of the large bowel down to a level of the junction of the ileum with the sigmoid, performing both operations at the same time. In order to avoid the removal of the colon and to reduce materially the risk of the operation, he effected a communication between the ileum and the large bowel in the true pelvis below the level of the last kink. This he found most successful. He believes that no one can become affected by tuberculous disease or by rheumatoid arthritis unless the resisting power of the individual to the entry of organisms has been depreciated by auto-intoxication consequent on the presence of chronic intestinal stasis. So in these conditions and especially in the case of progressive tuberculous affections of joints,

Lane employs no local operative procedures to the affected joint other than rest, and if necessary, aspiration of any fluid material that does not readily become absorbed after the operation; but proceeds to free the patient of the auto-intoxication by putting the ileum into the pelvic colon below the kink at the brim of the pelvis. In these cases the results of operation have been most successful, and the patient has improved both locally and generally. An obstruction develops frequently in the end of the ileum which plays an important part in the production of the group of symptoms which are regarded by the physician as evidence of indigestion. This plays an important part in the development of disturbances in the duodenum and stomach.

Infection of the Iliopsoas Muscle from Appendicitis.—MAUCLAIRE and AMAUDRUT (*Arch. gén. d. Chir.*, 1912, vi, 33) say that infection of the iliopsoas muscle, suppurative or non-suppurative, is a rare complication of appendicitis. Clerget collected 83 cases in which this muscle was infected. Of these, 13 were on the left side and 70 on the right side. Of those on the left side, one was caused by a perisigmoiditis, one by a salpingitis, one by an osteomyelitis of the pelvis, and one by a suppurative arthritis of the sacro-iliac joint. Of those on the right side, only 35 could be ascribed definitely to an appendicitis, while one or two more were doubtful. The others were due to direct trauma, puerperal infection, typhoid fever, or influenza. Mauclaire and Amaudrut report a case in which the infection of the muscle followed a mild appendicitis. Perforation of the appendix occurred in the second attack with a resulting infection of the muscle but not of the peritoneal cavity. Death followed operation, and although an autopsy was performed, the cause of death was not ascertained. It is concluded that while the condition is very rare it is grave. This complication seems to have some relation to an abnormal anatomical condition of the appendix, which after traversing an abnormal diverticulum situated under the iliac fascia, by its extraperitoneal extremity comes into direct contact with the psoas muscle. In the great majority of cases the appendix is in a retrocecal position. In 27 per cent. of the cases there had been one or more preceding attacks of appendicitis. The infection of the muscle can develop under two forms: One mild without suppuration, one grave with suppuration, which is more frequent than the other. In this form the local symptoms indicate the formation of an abscess, apparently tardy, toward the tenth or fourteenth day of the disease. The inflammation of the muscle is always accompanied by a grave general condition. Death occurred in 27 per cent. of the cases, notwithstanding operation, which should be done as early as possible.

Motor Insufficiency Due to Perigastritis of Gonorrheal Origin.—GREKOW (*Zentralbl. f. Chir.*, 1911, xxxix, 105) says that little attention has been paid to perigastritis, especially that which leads to impermeability of the pylorus and consecutive motor insufficiency of the stomach. The most frequent causes are associated with affections of the stomach and duodenum, as from ulcers and other affections. It may result from a focus in a distant organ as from perityphlitis, or diffuse peritonitis of various origins. Grekow believes that it can

result from tubal disease and that the gonococcus is particularly prone to produce it. He reports the case of a girl, aged eighteen years, who was admitted for a gonorrhea which was associated with a pelvic peritonitis. She was discharged July 20, 1909, with all symptoms improved except the occasional vomiting. She returned, however, two days later, with exacerbation of pain in the region of the stomach, and vomiting so frequent that the taking of food was impossible, and she was pale and thin. Her health previous to the beginning of the present trouble had always been good. No lues. No blood in the vomit or feces. Vomiting two or three times daily. Stomach dilated and palpable. Repeated tests showed no hydrochloride in the stomach contents. No tumor palpable. Operation, August 8, 1909, under local anesthesia. Stomach markedly dilated and lowered, and its walls hypertrophied. Numerous moderately loose adhesions in the region of the pylorus and gall-bladder. The omentum was loosely adherent to the right uterine adnexa, and contained enlarged and soft glands. In the remaining portions of the peritoneal cavity were found radiating adhesions. The pylorus had the appearance of a ring formed somewhat like a solid tumor. Neither the pylorus nor the duodenum showed any signs of a recent ulcer. No adhesions of the hepatic flexure of the colon, and the gall-bladder was free of stones or other changes except that its serous surfaces were rough and covered with a fine membrane. Appendix small and movable and unchanged. The adhesions were for the most part bluntly broken up. A posterior gastro-enterostomy with a Murphy button was performed, besides a gastroplication and gastropexy. The patient quickly improved and left the hospital well three weeks later. Three months after the operation she had a florid appearance, was free of symptoms, and had increased in weight 23.8 kilograms. This is a typical example of hypertrophy of the pylorus with dilatation of the stomach. The cause was the adhesions in the region of the pylorus which were undoubtedly due to the gonorrheal peritonitis. In a second case there was a diffuse peritonitis with dilatation of the stomach from rotation of the pylorus and duodenum as well as a slight hypertrophy of the pylorus.

The Formation of a New Bladder and a New Urethra.—Heitz-Boyer and Hovelacque (*Jour. d'Urolog. Med. et Chir.*, 1912, i, 237) say that the urine from the kidneys accumulate in a continent reservoir (the bladder), provided with an excretory canal (the urethra). These are accessible to instrumentation from without and every operation to correct a deficient reservoir should meet these requirements, whether the defect is the result of a congenital malformation (extrophy of the bladder), or of an accidental cause (total cystectomy, grave vesicovaginal fistula, etc.). Heitz-Boyer and Hovelacque conceived such an operation, which was put into practice by their chief, Marion, on a patient who nearly two years later was still in good health. The continence of the urine is assured by the anorectal sphincter which is subdivided into two passages; one fecal and the end of the intestinal canal, the other urinary constituting a new urethra leading from the newly formed bladder. The rectum is divided above within the pelvis. The upper segment is brought down and invaginated into the primitive anorectal canal, where it will occupy the greater part of the cir-

cumference of this canal, which has been denuded of its mucous membrane, in about the posterior three-fourths of its circumference. Both the upper and the lower segments of the divided rectum will make their exit at the site of the normal anus. The lower segment has been made to represent the bladder, the ureters being implanted into it. Both urinary and fecal canals are then separated, both are accessible externally to the cystoscope, and a valve-like septum exists between the two. The technique of the operation is given in detail and is well illustrated.

The Experimental Production of Basedow's Disease.—BIRCHER (*Zentralbl. f. Chir.*, 1912, xxxix, 138) refers to his previous publication of the results of experimental production of Basedow's disease, in which thymus gland tissue, as fresh as possible, from a thymus hypertrophy or from a patient with a persisting thymus, was implanted in the abdominal cavity. With other authors he had concluded that the thymus gland played an important part in the clinical picture of Basedow's disease, at least as important as that of the thyroid gland. The success of the thymus implantation in his cases was not surprising, he thought, in view of the mortality in the disease, the cure obtained, by Garre with thymectomy, and the symptoms produced by the secretion pressed from the thymus. Bircher is compelled to make this preliminary contribution because of recent antagonistic views. He intends to publish a more extensive paper later. He has produced in five dogs a complete clinical picture of the disease. The pieces of thymus were not obtained from patients suffering from Basedow's disease, but from those who died in shock from thymus persistence (2 cases), or from whom the thymus was removed on account of stenosis. The pieces were fresh and living, being in the air only about a half minute, and were implanted in the abdominal cavity. The results were decisive. In one dog (illustrated) a piece of thymus the size of the palm of the hand and 1 cm. thick, taken from an endemic cretin, was implanted in the omentum. The first symptoms developed in forty-eight hours. The dog was excited, sprang in his cage as if maniacal, took little food and showed much thirst. After four days protrusion of the eyes began to set in and reached its highest point on the twenty-eighth day. It remained stationary some days and then began to subside. It took five months to disappear. The palpebral cleft remained wide and the eyes could not be closed. The upper lid did not follow the eye in looking downward. On the third day tachycardia set in, and quickly reached 180 beats per minute. The jaws showed a rapid fine tremor which also affected somewhat the whole body. The goitre became palpable as a soft tumor after four or five weeks. The tachycardia persisted over three months and can in this dog still be recognized when he is excited. The other symptoms quickly disappeared. Two of the dogs experimented on were allowed to live, in order to study the disease further. Three were killed by a total thyroidectomy. These animals died with a marked tachycardia, with the picture of a quickly developing cachexia strumipriva. The symptoms of Basedow's disease were present in all five dogs in varying degree. Bircher considers that his experiments establish completely the relationship between the function of the thymus and Basedow's disease.

THERAPEUTICS

UNDER THE CHARGE OF

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The Treatment of Leukemia with Röntgen Rays.—BIERMANN (*Deut. med. Woch.*, 1912, xxxviii, 7) relates the details of 5 cases of leukemia treated by *x*-rays. One patient has remained apparently cured for eighteen months. This patient before treatment had a leukocyte count of 450,000, with a hemoglobin percentage of 55, and a red blood count of 2,600,000. Her blood picture and the marked enlargement of the spleen made the diagnosis clearly one of myelogenous leukemia. After the treatment the number of leukocytes was 12,500 and the spleen could no longer be felt. The general appearance of the woman was that of a perfectly well woman, although a differential count showed the presence of a considerable percentage of myelocytis. Biermann believes that myelogenous leukemia, especially in its early stage, is more amenable to *x*-ray therapy than other forms of leukemia. For this reason he emphasizes the importance of making an early diagnosis of leukemia. Biermann favors gradual and low dosage of the rays and notes briefly his method of procedure in applying this treatment to leukemic patients.

The Use of the Karell Diet in Heart Disease.—HIS (*Therap. Monats.*, 1912, xxvi, 10) mentions the indications for the Karell milk diet in various circulatory disturbances, and cites cases very favorably influenced by this method of treatment. The Karell "milk cure" is essentially a restriction to a strict diet of 800 c.c. to 1000 c.c. of milk a day, for a period of five or six days. This amount of milk does not, of course, furnish the necessary caloric value for the patient, and the treatment should be carried on only in bed patients. The indications are summed up under a number of different classes of patients: (1) His does not advise the usual Karell cure for cardiac disturbances in obese patients, but only the interposition of an occasional "milk day," as he believes that sudden cardiac weakness may occur if they are kept on too strict a diet for a long period of time. (2) Patients with chronic bronchitis and emphysema associated with increasing weakness of the right heart are much benefited by the Karell treatment. The dyspnea in such patients subsides in a few days on the Karell diet, and longer courses are seldom necessay. (3) Patients suffering from cardiac asthma with degeneration of the myocardium show a rapid improvement of all the symptoms that is often surprising, especially after heart tonics and sedatives have failed to relieve. After a week at most these patients can be allowed a light diet with a limitation of the fluids to 1000 to 1200 c.c., and if necessary the Karell diet may be repeated. (4) True angina pectoris is often remarkably benefited by

this treatment, especially the milder forms, in which the attacks are not severe but are frequently repeated. The results are especially good in plethoric patients with a tendency to meteorism, in whom the anginal attacks are incited by an overloaded stomach or by distended intestines. (5) Insufficiency of the kidneys is known to be benefited by a milk diet, especially when the heart shows signs of weakness, or if there are symptoms of impending uremia. (6) Valvular defects as such are no indication for this treatment, but when the heart muscle becomes insufficient and edema develops, much benefit is obtained by the limitation to the Karell diet. (7) Persistent effusions in the serous membranes often yield quickly to a Karell cure. Von Romberg has recommended the advantages of a salt-poor diet in tuberculous peritonitis. His believes that pleural and pericardial effusions disappear more readily than peritoneal effusions with this method of treatment. (8) This method is of great value as a supplement to digitalis therapy when neither alone is effectual He cites instances in which digitalis and other diuretics had no effect until after a period of restriction to the Karell diet. In one patient diuresis commenced on the sixteenth day after the restriction to milk, and in another case the diuresis began on the sixth day and did not reach the maximum until the twelfth day.

The Use of Subcutaneous Injections of Heroin in Cardiac Asthma.—FRAENKEL (*Therap. Monats.*, 1912, xxvi, 14) believes cardiac asthma is primarily due to a passive congestion of the pulmonary circulation. This congestion produces an overdistention of the alveolar capillaries, and consequently a diminution in the size of the alveolar spaces. As a secondary effect of the pulmonary congestion, swelling of the mucous membrane of the finer bronchioles occurs and the lumen of the finer bronchial tubes is often further diminished by spasms of the bronchial musculature. Fraenkel says that morphine acts very favorably upon all these causes of the dyspnea and hence its therapeutic effect in cardiac asthma is usually remarkable. Morphine, however, is open to the objection that it does not act so favorably when its use is long continued. Fraenkel recommends heroin as a substitute for morphine on the ground that heroin has the distinct advantage that it may be given daily for periods of weeks without losing its beneficial effects. The initial dose given by Fraenkel is 0.005 grams, and it may be increased to 0.015 grams. It is often of distinct advantage to combine the heroin with some form of digitalis or other cardiac tonics.

The Action of Iron in Chlorosis.—HEUBNER (*Therap. Monats.*, 1912, xxvi, 44) advances the hypothesis that in chlorosis the ability to reduce the higher salts of iron, the ferric salts, to the lower bivalent forms of iron, the ferrous salts, is lost. The iron in the food is in the form of the higher salts, and chlorotic subjects are not able to assimilate it. He believes that the empiric use of Blaud's pills, of syrup of ferrous iodide, and certain natural mineral waters that contain iron in the bivalent form is justified upon the basis of his hypothesis. The clinical fact that these preparations are particularly useful in the treatment of chlorosis tends to strengthen the theory as advanced by Heubner. On the other hand, the therapeutic effects of iron preparations, such as ferratin, ferrum, oxydatum saccharatum, and hundreds of pro-

prietary preparations have often been disappointing. This, according to Heubner, is due to the fact that these are all the higher salts of iron, and the chlorotic subject is incapable of reducing the iron to a form suitable for absorption.

The Effects of Subdural Injections of Leukocytes on the Development and Course of Experimental Tuberculous Meningitis.—MANWARING (*Jour. Exper. Med.*, 1912, xv, 1) says that cerebrospinal membranes differ from the true serous membranes in that they are almost impervious to blood proteids and normally receive little or no protective substance from this source, and few, if any, leukocytes. Therefore, it becomes necessary to treat pathological processes existing within these membranes by direct inoculation of remedial agents subdurally or into the spinal canal. The author relates his method for producing expermental tuberculous meningitis in dogs and gives a brief summary of the results of subdural inoculations of tubercle bacilli of varying virulence. The leukocytes used in treating the infected animals were obtained by the injection of turpentine into the pleural cavity of dogs, as perfected by Opie in his study of the treatment of experimental tuberculous pleurisy. Manwaring found in his experiments that subdural inoculations of tubercle bacilli of established virulence for guinea-pigs and rabbits produces in the dog a tuberculous meningitis followed by paralysis and death. When suspensions of canine leukocytes are injected subdurally, following such inoculations there occurs uniformly a delay in the development of the paralysis and a prolongation of the life of the treated animal. In dogs inoculated with small doses of tubercle bacilli of low virulence, the development of paralysis has been prevented by this means for periods of seven months (up to the present date), while the untreated animals injected with the same cultures have all developed paralytic symptoms within a period of about four weeks.

Chemotherapeutic Experiments on Animals Suffering from Malignant Tumors.—A. WASSERMANN, HANSEMANN, KEYSER, and M. WASSERMANN (*Berl. klin. Woch.*, 1912, xlix, 4) relate their experiments in an endeavor to find a chemical substance capable of exerting a specific destructive action upon the cells of malignant tumors occurring in mice either spontaneously or as a result of inoculation. They found in experiments in vitro that selenium and tellurium salts were taken up selectively by carcinoma cells. They also found that when salts of selenium and tellurium were injected locally into the tumors of mice affected with carcinoma these tumors softened and liquefied. However, it is not possible to inject all malignant tumors locally, and any attempt to cure a malignant tumor must take in consideration the possibility of reaching all the tumor cells that possibly may be distributed by metastases widely throughout the body. They next attempted to find some chemical preparation derived from these metals that could be injected into the blood, and that would distribute itself rapidly throughout the body and at the same time be diffusible. More than 200 chemical combinations were tried before Wassermann and his co-workers obtained a preparation consisting of a compound of eosin and selenium. This chemical combination is unstable, and the exact

nature of it is not given. This substance is a red powder very easily soluble in water. After injections of this solution into the caudal vein of mice there occurs almost immediately a diffuse reddening of the entire animal that usually disappears within twelve hours. The injections were given daily and after the third injection a distinct softening of the tumor was evident on palpation. After the fourth injection this softening was more evident and the formerly solid tumor now appeared to be a fluctuating cyst. The injections were continued, and usually in ten days a complete disappearance of the tumor was noted. The injections, however, did not always lead to this favorable result, as many of the animals died after the first, second, or third injection. This result may have been due to improperly prepared preparations. Mice with large tumors frequently become severely poisoned when liquefaction and softening of the tumor occurred rapidly. The authors are of the opinion that these toxic symptoms are due to the absorption of the destroyed tumor. The animals in whom the tumors healed have remained without recurrence for months. They conclude from these experiments that it is possible to cure malignant tumors in mice by this means provided the tumors are not too large in proportion to the body weight of the animal. They wish to warn that there is no evidence that eosin-selenium will act similarly with human cancer. However, their experiments do point out a line of investigation that may be extended and lead to a progress in human therapy.

Hexamethylenamin in the Treatment of Bronchitis.—VANDERHOOF (*Jour. Amer. Med. Assoc.*, 1912, lviii, 331) recommends hexamethylenamin as a remedy of great value in cases of acute colds and in patients suffering from acute and chronic bronchitis. The drug should be given in large doses accompanied by the copious drinking of water. In the ordinary cold, treatment with hexamethylenamin shortens the stage of coryza and greatly modifies or entirely prevents the succeeding bronchitis. He also believes that it acts as a prophylactic against ensuing infection of the accessory nasal sinuses. Vanderhoof says that it is our best remedy in acute bronchitis. Certain cases of chronic bronchitis respond to treatment by hexamethylenamin with a gratifying alacrity, while others do not. In the latter instance it is presumed that structural changes have occurred in the walls of the bronchi, associated with thickening and calcification of the cartilages, fibrous membrane, and muscular coats so as to preclude the hope of successful treatment by any remedy.

Salvarsan in Syphilis of the Nervous System.—SACHS and STRAUSS (*Med. Record.*, 1912, lxxxi, 211) have used salvarsan in 80 cases of syphilis of the nervous system, and conclude that salvarsan has no curative effect in tabes and general paresis. Salvarsan seems to influence favorably some of the symptoms particularly the vesical and sexual functions, and under its use some of the cases remained stationary. None of the patients treated with salvarsan exhibited any unfavorable symptoms attributable to the drug itself. They believe that in these two diseases it accomplishes as much as, but not any more than was achieved in former years by the use of mercurials. In brain and

spinal cord syphilis the use of salvarsan is followed by an improvement in many of the symptoms, but they do not believe that the results obtained with it have been better than those obtained by mercury. In the acute and subacute form of brain syphilis, especially when associated with convulsive seizures and with chronic headaches, the drug has been distinctly beneficial. Without doubt the good effects of this treatment may be made more lasting by the additional use of mercury and the iodides. Although their results were negative with its use as a therapeutic agent, yet they hope that the use of salvarsan in the early stages of syphilis may prevent the development of late and more serious forms of specific disease of the central nervous system.

Personal Experiences with Digipuratum.—BREITMAIER (*Deut. med. Woch.*, 1911, xxxvii, 2376) recommends digipuratum as a substitute for digitalis extracts and tinctures. He believes that it is much less apt to vary in strength than other digitalis preparations, and that it does not deteriorate with age. He believes that the absorption and elimination of digipuratum are more rapid than other digitalis products, and therefore cumulative action of the drug is less likely to recur. Digipuratum also causes less digestive disturbances, due to the fact that certain irritating elements that occur in other digitalis preparations have been removed. It has the additional advantage that it can be given intravenously.

Digitalis Preparations Employed in Medicine.—DIXON (*Quart. Jour. Med.*, 1912, v, 297) reviews the findings of many observers on the action and effects of the different digitalis preparations employed in medicine. The three principal glucosides of digitalis leaves are digitoxin, digitalin, and digitonin. He describes the properties of these three glucosides briefly. Digitonin is a saponin found principally in digitalis seeds and has no value as a cardiac tonic. It is soluble only with difficulty in cold water but is much more soluble mixed with digitalin. It is owing to the presence of digitonin in digitalis leaves that digitoxin and digitonin find their way into the watery preparations, such as the infusion. Digitalin has an effect on the heart similar in the main to digitoxin, but is only about half as active. Digitoxin is the most active constituent of digitalis leaves. Its insolubility renders it difficult to deal with; it is very irritant, and if given by the mouth causes gastric symptoms more than any other digitalis preparation. The subcutaneous administration of digitoxin causes severe pain, and it may induce necrosis and suppuration. It is slowly absorbed and is still more slowly eliminated, and therefore has a cumulative effect. One great object in preparing substitutes for the galenical preparations of digitalis has been to obtain a form suitable for subcutaneous injections. Another objection to the use of the ordinary preparations of digitalis is the fact that the therapeutic effects, especially the diuresis, do not appear before the second day, and more commonly on the third day, and this renders the drug of little value in acute conditions, and when an immediate action is required. Dixon describes briefly the three most important proprietary preparations which have been introduced to meet the disadvantages of the ordinary digitalis preparations. Digalen or soluble digitoxin was first prepared by Cloetta, who

claimed for it a superiority over digitoxin, in that it was soluble in water, less irritant to the stomach, more constant in strength, more rapid in its action, and, lastly, that it was not cumulative like digitoxin. Dixon cites the widely opposed views of different observers regarding these advantages claimed for digalen. Clinicians vary in the widest degree in reports as to its clinical value. Some think it is an ideal digitalis preparation, while others regard it as most unsatisfactory. Digalen is probably not so irritant to the stomach and alimentary canal as digitoxin. Subcutaneous injections of digalen are very painful and frequently produce inflammatory reactions. The consensus of opinion is that it is not more rapid or constant in its action than a properly standarized tincture and has also a distinct cumulative action. Digipuratum is a preparation made from digitalis leaves by exhausting them with alcohol and ether. By this means it is stated that 85 per cent. of the inert material in digitalis leaves, including the saponins, is removed and that the remaining 15 per cent. contains all but a small percentage of the active constituents. The four virtues which can be assigned to digipuratum are that it keeps well, it is uniform in composition, it contains all the glucosides of value in digitalis, and causes less gastric irritation than the powdered leaves. The one great disadvantage is that it is ten times more expensive than the tincture of digitalis, and Dixon says that a properly standardized tincture of digitalis has all the advantages of digipuratum. Clinical reports on digipuratum are on the whole very favorable. Digitalone, like digalen and digipuratum, has a typical digitalis action on the heart, and it is claimed for this preparation by the manufacturers that it is suitable for oral, hypodermic, intravenous, or rectal administration. Dixon does not believe that the action of digitalone differs from the ordinary galenical forms except that it is less active. In concludion, it may be stated that pharmacological and clinical investigations have not made a clear case for the substitution of active principles for galenical preparations of digitalis in the treatment of patients. These active principles are for the most part just as irritant as the infusion or tincture; they are neither more reliable and constant in action than a properly standardized tincture, nor are they absorbed more rapidly; they have the same tendency to accumulate and are much more expensive. The one advantage that certain of them may possess over the pharmacopœal preparations is that they may be given intravenously without ill effects.

The Treatment of Syphilitic Heart and Vascular Disease.—Oigaard (*Zeit. f. klin. Med.*, 1911, lxxiii, 440) says that the Wassermann reaction is of great value as an aid to the diagnosis of syphilitic aotitis that is in many cases difficult to diagnosticate clinically. Aneurysm of the arch of the aorta and aortic insufficiency are almost always of syphilitic origin and the Wassermann reaction is usually positive. Mercurial treatment was given to the patients reported by Oigaard with very good results. He also tried iodides but does not believe they have any effect upon the course of the disease. The mercurial treatment should be continued until the disappearance of all subjective symptoms, and until the positive Wassermann reaction becomes negative. Treatment should be resumed upon the recurrence of any subjective symptoms or a return to a positive Wassermann reaction.

PEDIATRICS

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The Therapy of Tuberculous Ascites in Children.—MAX KLOTZ (*Berl. klin. Woch.*, 1912, xlix, 73) points out that the withdrawal of the ascitic fluid by paracentesis in tuberculous peritonitis, is not always dangerous or useless. Out of 11 cases he mentions 5, in 2 of which a single tapping was sufficient to permanently cure the ascites. Two cases required two tappings to accomplish this, and 1 case required four tappings. The indications for tapping in this condition are, when there is an increase in, or no decrease of, the ascites after rest in bed, dieting, sun baths, food poor in salt and diuretics. If the fluid returns another tapping is indicated. If there is no return of fluid in fourteen days the patient is allowed to sit up one hour at a time, gradually increasing the periods. Klotz has seen no bad effects occurring from this plan of allowing the patient to sit up, although a number of authorities make a point of long continued rest in bed. Klotz has seen the return of the ascites under this latter method. The length of the treatment varied from five to nine months. Five out of 11 cases are clinically cured or well after nine to twelve months from their discharge. One child had an exudative pleurisy complicating the ascites. Tuberculin, tried in 2 cases, showed some improvement in 1 case, and had no effect in the other. The acholic stools which Berggrun and others hold as of diagnostic value were not seen in any case of tuberculous ascites. Infiltration of the umbilical region, which Schmidt found in 7 per cent. of his cases, is rare, and occurred but once in the 11 cases of Klotz. Klotz claims that in general practice good results can be obtained by any physician who employs the hygienic and dietetic treatment given above and performs paracentesis under the above conditions and rules.

The Prognosis in Epilepsy in Children.—JULIUS ZAPPERT (*Med. Klinik.*, 1912, viii, 229) calls attention to the difficulty of prognosis in the epilepsy of children, a condition in which the diagnosis alone offers great difficulties. Besides excluding hysterical attacks and the convulsions of childhood there remain doubtful cases with pronounced epileptiform convulsions which cannot be pronounced true epilepsy. Also in cases giving the picture of uncomplicated true epilepsy, a subsequent change in the symptom-complex is possible, so that the rule may be laid down that in childhood these cases should be diagnosticated as epileptiform attacks and not as true epilepsy. While a large number of cases with typical attacks in early childhood subsequently develop severe and permanent epilepsy, there remains quite a number of cases in which typical attacks occurred frequently for from one to three years and then diminished and disappeared altogether without subsequent return to date. Zappert gives short histories of 7 such

cases. These cases were almost invariably boys and the attacks were typical of epilepsy. Another type exhibits typical attacks associated with reflex irritation from intestinal parasites, the attacks ceasing with the discharge of the parasite. Again, in cases of so-called spasmodic or idiopathic eclampsia in childhood, Birk and others claim that a certain number develop true epilepsy in adult life, while Hochsinger claims there is no genetic relation between the two conditions. Zappert details a number of cases which gave typical attacks associated with symptoms of pseudobulbar palsy. These attacks lasted from one to two years and the prognosis was bad. However, under treatment with bromides the attacks ceased and have not recurred. Here we have the possibility, however, of their reappearance at some future time. In the prognosis of epilepsy it must be remembered that severe and incurable diseases of the brain often stand back of the attacks. There are many cases in which the diagnosis of epilepsy can be made with a high degree of certainty, and which, even after sedative treatment is stopped, show improvement and an absence of symptoms for one or more years. It seems proper to offer a milder prognosis than heretofore in the epilepsy of childhood, and hold out at least the probability of a cessation of the attacks for a long time, and the possibility of a spontaneous cure.

The Influence of Heredity on the Development of Cardiac Disease.—Max Herz (*Münch. med Woch.*, 1912, lix, 419) believes that more weight should be placed on the influence of heredity on the development of cardiac conditions. The results of this influence in a number of cases has been of great significance in the differential diagnosis, and especially in the prognosis, of cardiac conditions. Herz briefly outlines from his experience the influence of heredity in the three chief groups of cardiac affections, namely, the neuroses, the rheumatic, and the arteriosclerotic conditions. The simultaneous appearance of a nervous cardiac affection in parents and their children is a common observation. Especially frequent is a nervous extrasystole in children of neurotic parents. The frequent occurrence of articular rheumatism, chorea, and the attendant cardiac lesions in families is well known. A predisposition for this condition cannot be doubted. In the majority of cases the child inherits this predisposition from the mother. The above facts become of significance in determining whether a cardiac murmur in a child is due to valvular disease or not. In doubtful cases it is better to accept as functional a cardiac murmur in a child between six years and puberty, since experience shows that in most cases the murmur will disappear during puberty. However, if the child's mother presents the stigmata of a rheumatic constitution, the prognosis should be decidedly more guarded. The clearest and most impressive influence of heredity, however, is found in the condition of sclerotic cardiac disease. Sudden death from cardiac failure at a certain age is especially common in some families. This occurs usually from the fortieth to the sixtieth year. In connection with nervous cardiac affections the element of imitation or mimicry is a factor not only in cases of children imitating the symptoms of cardiac neuroses in their parents, but also in cases of susceptible attendants or nurses in cases of organic cardiac disease.

OBSTETRICS

UNDER THE CHARGE OF

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The Pathogenesis of Hemorrhage in the Newborn.—BONNAIRE and DURANTE (*L'Obstétrique*, October 10, 1911) report 4 cases of fatal hepatic hemorrhage, non-traumatic, in newborn infants, accompanied by considerable bleeding into the peritoneal cavity. They analyze these cases in detail, and come to the conclusion that under the influence of an intoxication or a chronic infection in the mother, vascular lesions and cellular degeneration in the liver develop in the infant, diminishing the formation of fibrinogen and causing a deficient power of coagulation in the child's blood. At the moment of birth the intoxication or infection produces a polyglobular condition in the infant, with excessive leukocytosis and congestive phenomena. The blood-vessels are diseased and the capillaries altered by the cellular degeneration in the parenchyma of the organs. In a child previously healthy the traumatic lesion producing rupture of the vessels might end in recovery; but in these cases hemorrhage occurs in the parenchyma of the liver or beneath Glisson's capsule, causing distention and the final extravasation of blood in the peritoneal cavity. The reviewer has recently seen two cases of this condition. In one, a comparatively young woman, there was chronic catarrh of the bile ducts, and when in the non-pregnant condition she suffered repeatedly from biliousness and jaundice. After easy parturition she lost two apparently well-nourished infants, the first from intestinal hemorrhage; the second from diffuse pulmonary hemorrhage. In the latter a thorough autopsy demonstrated multiple hemorrhage into the parenchyma of the liver, and a bacillus was isolated from the blood and tissues of the infant. The mother has been urged to submit to a cholecystotomy and thorough drainage of the gall-bladder. This advice has not been accepted, but she has compromised by a Carlsbad cure. The second case, a primipara, older than the average, disregarded hygienic precautions in her diet during pregnancy, and lost her infant from hepatic bleeding.

Postoperative Thrombosis and Embolism.—KLEIN (*Monats. f. Geburts. u. Gynäk.*, 1911, Band xxxiv, Heft 5) gives the results of his studies on this question over a period of twelve and one-half years. Most of the cases studied were those of gynecological operations, although there were among these some cases of general surgery, such as extirpation of the breast for carcinoma, operations for hernia, appendectomy, and other operations on the intestine. Cases of confinement and the puerperal period are not included in this report, and cases of endometritis preceded by abortion are also omitted. The total number of operations is 5524, with 50 cases of thrombosis and embolism, *i. e.*, 0.9 per cent. In 1720 laparotomies there were 29

cases of thrombosis, or 1.7 per cent. The former belief was that thrombosis and embolism depended upon the nature of the patient's illness and the sort of operation. More recent knowledge indicates that thrombosis is dependent upon physical reasons, and that alteration in the blood and slowing of the circulation are important factors in the case. Such diseases as uterine myomata, often accompanied by degeneration of the heart muscle, predispose to this accident. It is also true that the more serious the operation, the greater the liability, and that more cases of laparotomy have this accident than of less severe operations. Thrombosis and embolism is most frequent after abdominal section for the removal of myomata. In comparing abdominal section with vaginal section, he found, in 1720 abdominal sections, that there were 29 cases of thrombosis, or 1.7 per cent.; while in 1992 vaginal sections there was but 0.8 per cent. of thrombosis, or not quite one-half in abdominal section for myomata. Whether supravaginal amputation or total extirpation was performed the number of cases of thrombosis was three times as great as after vaginal operations for the removal of myomata. The most serious result of thrombosis is fatal embolism. In the 1720 abdominal sections there were 9 cases of fatal embolism; while in 1992 cases of vaginal section there was none. The vaginal operations were quite as severe as those performed through the abdominal wall. There seems to be an important advantage in the choice of the vaginal method of operating. Thrombosis after operations on the adnexa is not rare. Most of these cases were not those of inflammatory disease, but of primary tumor of the tubes or ovaries. In 1905 operations upon the adnexa, there were 11 cases of thrombosis, or 1 per cent., and 2 fatal cases of embolism. One of these was after removing a carcinoma of the left ovary. After a radical abdominal operation the patient did well until the ninth day, when thrombosis in the left leg appeared, followed shortly after by fatal pulmonary embolism. The patient was extensively diseased in many ways. The second case was that of a woman aged forty-eight years, operated upon for hydrosalpinx and cystic ovary of the right side. On the fifth day after matters were apparently going well, there was fatal pulmonary embolism. Thrombosis was rare after operations for prolapse and retroflexion, but after curetting for hemorrhagic endometritis the accident occurred on the fifth day. When the age of the patients is considered, 52 per cent. of all cases were in middle life, between forty-one and fifty-one years. The youngest woman was aged nineteen, the oldest sixty-seven years. In 38 cases one could detect the beginning of the thrombosis, and the greater number happened between the first and tenth days after operation; and an almost equal number between the eleventh and twenty-first days. Most of the cases occurred in the first three weeks after operation. The majority of the cases occurred on the left side of the pelvis and in the veins of the left thigh. In 44 patients there were 24 cases on the left, 11 on the right, and 9 on both sides. Most cases of embolism are pulmonary, and in 13 cases 10 were of this nature. There was one apoplexy, where after thrombosis of the left crural and iliac veins an embolus found its way to the fissure of Sylvius. These cases seemed more common after operations for carcinoma. In classifying operations, every case in which the uterus was extirpated was considered a

radical operation. One of these, followed by fatal embolism, was done for carcinoma. In 41 radical operations for carcinoma there were 16 abdominal sections, and 25 vaginal sections, in a total of 563 operations for carcinoma; 0.2 per cent. of these had thrombosis. In searching for a cause for this accident, one naturally turns first to the possibility of an infection in the blood. If we are to diagnosticate this by a rise in temperature, such a condition was absent in 14 cases, which is 28 per cent. of all the cases of thrombosis. Direct infection of the wound could be traced in 8 cases, or 16 per cent. In these cases there was either abscess in the abdominal wall, or suppuration in the vaginal wound. Other causes are those mechanical factors which disturb greatly intra-abdominal pressure, such as large tumors in the pelvis in 7 cases, ascites in 3 cases; marasmus and anemia in 1; pernicious anemia in 14; insufficiency of the heart's action in 12; arteriosclerosis in 2; bruising of the vessels by mass ligatures or resections of large veins, in 4 cases; prolonged anesthesia in 6; varices in 4; and other conditions affecting the general health. All of these tend to alter the constitution of the blood, disturb blood pressure, and retard circulation. To avoid this accident one should guard carefully the strength of the heart muscle, if necessary giving digitalis for some time before operation, and when circumstances are favorable avoiding delay, because of its depressing influence. It is thought that lumbar anesthesia is less dangerous than that by inhalation. Strict aspesis, avoidance of mass ligature, and wounding of the large veins should be observed, and, if possible, the avoidance of chill on the skin of the abdomen. After operation the patient should receive nutritious foods in great abundance, and if necessary the circulation should be promoted by massage or passive movements. The comparative frequency of this accident is studied by quoting the reports from various clinics, aggregating between 30,000 and 40,000 operations. The frequency of the accident varied from 3.6 per cent to 1 per cent.

The Sterilization of the Skin.—LEEDHAM-GREEN (*Brit. Med. Jour.*, October 28, 1911) has made culture experiments with various strengths of iodine, with alcohol, and with sublimate alcohol, in sterilizing the skin. Although a final conclusion has not been reached, the experiments so far may indicate a slight superiority in sublimate alcohol over iodine, than alcohol alone. When sublimate alcohol and iodine were merely painted on the skin there was little difference in the result; but when sublimate alcohol was thoroughly rubbed into the skin it proved in the majority of cases more efficient. The sublimate solution used was 1 to 1000 in 70 per cent. alcohol. This was compared with 70 per cent. official tincture of iodine and 1 to 1000 aqueous solution of bichloride of mercury. The result was in favor of the 70 per cent. sublimate alcohol.

GYNECOLOGY

UNDER THE CHARGE OF

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Anatomical Cure of Cystocele.—WHITE (*Amer. Jour. Obst.*, 1912, lxv, 286) does not believe that any of the commonly accepted theories as to the pathological anatomy of cystocele are correct, and thinks that the unsatisfactory results obtained in its treatment are due in large measure to a faulty conception of the cause. His studies on the cadaver have convinced him that the bladder stays in place because it rests on a firm fibrous shelf, which stretches across between the pubic bones from the symphysis to the spines of the ischium, this shelf being nothing more than the anterior vaginal wall, which is attached by firm adhesions to the pubic bone in front, and laterally to the whole length of the white line of the pelvic fascia. This can be easily demonstrated by running a knife along the white line on either side and severing the attachments to the vagina; a cystocele of marked degree will be produced. Upon suturing the vagina back to the white line, normal conditions are restored, and the cystocele is cured. Acting upon this theory, White has devised the following operation. An incision is made in the anterior lateral fornix of the vagina, extending from the level of the cervix to near that of the internal meatus of the urethra. The finger is then worked by blunt dissection toward the side of the pelvis until it can be placed on the uncovered ischiadic spine. By means of a curved needle three or four sutures are then passed through the lateral edge of the vaginal incision, around back of the white line, and out through the mesial edge of the vaginal incision; when these are tied the lateral fornix of the vagina is drawn up into contact with the white line. The other side is treated in the same manner, so that at the completion of the operation the anterior vaginal wall stretches across from one ischiadic spine to the other, reëstablishing the normal shelf-like condition. The operation sacrifices no tissue, and in the author's experience has given most satisfactory immediate and remote results.

Etiology of Appendicitis.—Excessive eating, especially of red meats, is an important factor in the causation of appendicitis, according to DICKINSON (*Amer. Jour. Obst.*, 1912, lxv, 284), who calls attention to the fact that man is the only animal that suffers from this disease, and that he is the only one that makes eating a pleasure, filling his intestinal tract with high proteids, the undigested portions of which undergo fermentation. It is also worthy of note in this connection that physicians working in China, where red meat is very little eaten, report rarely seeing a case of appendicitis among thousands of patients examined. Dickinson believes, therefore, that there is a close connection between gluttony and the disease, but also lays emphasis on the

importance of the condition of drainage in the appendix in determining the type. He distinguishes between the progressive, destructive form, and the chronic hyperplastic, which goes on to fibrosis. In the former the ceco-appendicular junction is tubular, the lymphoid tissue at the junction being so swollen as to choke the aperture, so that the appendix cannot drain itself; this results in tension, leading to gangrene or perforation. In the chronic or hyperplastic type the ceco-appendicular junction is funicular, embryonal in character; drainage is good, and tension does not occur. The sequence of events is therefore probably somewhat as follows: overeating of high proteids, residuum in cecum, decomposition, ceco-appendicitis. The cecum, draining well, recovers; the appendix, if not draining at all, goes on to destruction; if draining poorly, to subacute appendicitis with hyperplasia; if draining well, to chronic appendicitis with fibrosis.

Conservative Surgery of the Ovaries.—Dickinson (*Surg., Gyn., and Obst.*, 1912, xiv, 134) has studied the results in a series of 131 operations performed on intelligent women from private practice. All these were done more than six months ago, this arbitrary time-limit having been chosen because symptoms of the surgical menopause generally develop within this time if at all. In 80 per cent. of the cases in which one or both ovaries were left, no climacteric disturbances of any kind occurred. The failure to prevent these entirely in the remaining fifth of the cases may have been due, Dickinson thinks, to lack of skill or care in preserving the ovarian circulation. He believes that this is better accomplished by leaving the tubes as well, when they are healthy, as important nerves and arteries going to the ovary may be cut in removing them. His doctrine is that all healthy ovaries should be left in place, even if the patient is near the menopause, since no one can say just what is the term of ovarian activity, and especially is this true in fibroid cases, in which the ovary is notoriously long-lived. Dickinson believes that better results are obtained when both ovaries are left than when one is removed, or resections are done. He has found that, as far as can be determined, conservation in married women is followed by persistence of sexual desire in almost all cases. The question of leaving or removing ovaries in operations for advanced inflammatory disease is often a very difficult one to decide, as these ovaries are apt to be very tender, and to give rise to trouble later; Dickinson says that he is, therefore, rather inclined to remove them under such circumstances, unless the patient is fairly young and vigorous, and there seems to be no infection of the ovary.

Post-climateric Metrorrhagia of Non-cancerous Origin.—Dalché (*Gaz. des Hôp.*, 1912, lxxxv, 3) calls attention to the fact that not all uterine hemorrhages occurring after the menopause are due to malignancy, and that although we should never loose sight of the overwhelming importance of this etiology, neither should we overlook the fact that certain of these cases are amenable to medical treatment, or undergo spontaneous cure. Sufferers from an old metritis, following frequent childbirths, and accompanied by a general visceroptosis, are frequently the subjects of severe post-climacteric hemorrhages. These may likewise occur, often associated with a profuse, fetid discharge,

as a result of senile metritis, the condition clearing up entirely after a simple dilatation of the cervix and intra-uterine lavage. Mucous polyps and fibromyomas are well recognized causes of post-climacteric bleeding, which may be very severe and intractable even in cases of but a single small submucous nodule. A condition, however, which in Dalché's opinion is not thought of nearly often enough as the etiological factor in these cases is syphilis. This may manifest itself as a diffuse syphiloma, forming a large uterine and peri-uterine mass, as multiple gummas, forming a friable, nodular mass on the cervix, or merely as a sclerotic condition of the uterus and its vessels, all these conditions giving rise to metrorrhagias, and all being cured by specific treatment. It should not be forgotten, moreover, that post-climacteric bleeding may in some instances be due to an actual return of menstruation, corresponding to a tardy ovulation. Finally, such hemorrhages may be due to extragenital causes altogether, such as endo- and myocarditis, hepatic or renal diseases, lumbo-abdominal neuralgia, and obesity. All these conditions may be accompanied by metrorrhagia, whose cause is to be sought outside of the genital sphere.

True Bone Formation in the Tubes and Ovaries.—Four cases of this exceedingly rare condition are reported by Pozzi and Bender (*Rev. de gyn. et de chir. abdom.*, 1912, xviii, 129), 2 of these affecting the tube and 2 the ovary. After a most careful analytical study of the literature Pozzi and Bender were able to find but 5 additional cases of true ossification in the ovary, and but 2 additional cases in the tube. They conclude from the study of their own cases and of those from the literature that the process is identical, whether its seat be in the ovary or the tube. These studies have shown the fallacy of the older theory that development of osseous tissue in the ovary is inseparable from the existence of a teratoma or dermoid cyst, as there were no traces of anything of the sort in their specimens. What is the significance of this intrinsic, true ossification of the ovary and tube? It is not a neoplastic process, hence the term "osteoma" is inappropriate. Pozzi and Bender believe that the osteogeneiss, which is always decidedly limited in extent, is a regressive modification of preexisting tissue elements. The ossification is always preceded by calcification. Histologically actual bone tissue was found in all the cases, with osteoblasts and marrow tissue, the latter containing all the cell-types found in normal marrow. In no case was cartilage found, so that the ossification cannot be considered as having occurred from aberrant cartilage rests. In some instances a direct metamorphosis of fibrous tissue into bone had occurred, such as is seen in the formation of the membrane-bones of the skull; in other cases calcification had evidently taken place, followed by irritation, this causing proliferation of connective tissue, with the formation of myeloid tissue containing osteoblasts, these then causing the bone formation. According to these observations bone formation in the tubes and ovaries, although occurring very rarely, follows exactly the same laws that govern heterotopic osteogenesis in other portions of the body.

HYGIENE AND PUBLIC HEALTH

UNDER THE CHARGE OF
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The Influence of Sterile Environment, Inspired Air, and Food on the Digestion and Metabolism of Animals.—KIANIZIN (*Jour. de Physiol. et de Path. gén.*, September 15, 1911, xv) placed experimental animals in a sterile bell jar through which sterilized air was circulated. The sterilization of the air was controlled by drawing it, before its passage into the bell jar, through sterile broth. The broth remained clear throughout the experiments, so that there is little doubt but that the air was freed of microörganisms. The animal under observation in the bell jar was placed on a shelf composed of close-meshed wire netting. The excrement dropped through this netting into a strong solution of boric acid. The urine was collected separately. The total nitrogen and the relation of the nitrogen of the urea to total nitrogen was studied, and the ratio of the nitrogen of the urea to total nitrogen was taken as an index of the metabolism. These experiments in each instance showed a reduction in the ratio between urea nitrogen and the total nitrogen, but this reduction in no instance was very considerable. Folin has shown that this ratio between the nitrogen of the urea and the total nitrogen may undergo considerable variation in normal adults upon a mixed diet, so that the relatively slight changes in the nitrogen ratio mentioned by Kianizin are not to be regarded as proving absolutely that bacteria, which ordinarily are present in the air and the food, are absolutely indispensable for the life of the organism. It should be stated that many of the experimental animals died rather quickly. Kianizin did not believe that the cause of death in these cases was due wholly to digestive troubles; he believed that they were caused by an intoxication due to modifications of metabolism. The experiments, while very suggestive, are not conclusive.

The Hygiene of the Swimming Pool.—JOHN W. M. BUNKER (*Amer. Jour. Pub. Hyg.*, November, 1910, xx,) and WHIPPLE and BUNKER (*Eighteenth Annual Convention of the American Society of Municipal Improvements*, 1911) have studied the effects of "bleach" (calcium hypochlorite) in purifying the water of swimming pools. Chemical analyses have shown that the water of swimming pools becomes polluted, the degree of pollution depending largely upon the number of persons bathing in the pool. This pollution is shown by an increase in the amount of free ammonia, albuminoid ammonia, and by a decided increase in the number of bacteria. It is a curious fact that the numbers of Bacillus coli are not increased as markedly as might be expected. In a series of experiments, which are recorded, the colon bacillus is

uniformly absent in 0.1 c.c. of water from the swimming pool even after ten days, but is irregularly present in 1 c.c., and quite generally present in 10 c.c. In one experiment the bacteria growing on gelatine at 20° C. increased in numbers from 205 per cubic centimeter to 255,000 in three days, and to 281,000 in four days. After using "bleach" in the proportion of 1 part per million, these numbers were reduced very materially. Thus, a sample of water obtained from the new swimming tank in Brown University showed an original bacterial content of 700 c.c., and this was reduced to zero in fifteen minutes following the addition of the hypochlorite. A similar experiment carried out in the tank itself gave somewhat similar results. Surface samples before the addition of "bleach" yielded a count of 500 bacteria per cubic centimeter. Fifteen minutes after adding one-half a part of bleach per million of water these bacteria were reduced from 700 to 30 per cubic centimeter, and after thirty minutes to 10. At the end of an hour the water was sterile (*i. e.*, no colonies on plates). A sample taken eight hours later, while the pool was still agitated from those who had bathed in it, showed a bacterial content of 5 per cubic centimeter. The pool remained practically sterile for four days, at the end of which time the bacterial count began to rise steadily. It would seem that the application of hydrochlorite of lime offers a cheap, efficient, and convenient method of keeping the number of bacteria in a swimming pool down, and this diminishing the potential danger incident to bathing in a common tub.

The Etiology and Transmission of Measles.—ANDERSON and GOLDBERGER, in a series of articles (*Public Health Reports*, June 9, 1911, xxvi, 847; *Ibid.*, 887; *Jour. Amer. Med. Assoc.*, July 8, 1911, lvii, 113, 114; *Ibid.*, August 5, 1911, lvii, 476 to 478; *Ibid.*, 971, 972; *Ibid*, 1612, 1613) have reported the results of their experiments upon the etiology and mode of transmission of measles. In the first paper by these authors they report upon the susceptibility of the rhesus monkey to inoculation with blood from human cases of measles. Previous to their work it had been generally accepted among laboratory workers that monkeys were not susceptible to the disease. In their third paper Anderson and Goldberger show that the apparent non-susceptibility of the monkey to infection with measles was due to a more or less limitation of the period of infectivity of the blood, beginning at least several hours before, and continuing for about twenty-four hours after, the first appearance of the eruption. At the end of twenty-four hours from the first appearance of the eruption the infectivity of the blood for the rhesus monkey becomes greatly lessened and becomes progressively less thereafter. They draw attention to the fact that this limited period of infectivity also throws much doubt upon the value of previous bacteriological studies of the blood in measles, as it is quite possible that many workers have used non-infective blood. The fourth paper is on the nature of the virus existing in the circulating blood. They found that in a certain proportion of cases the measles virus in the blood is capable of passing through a Berkefeld filter. It resists drying for at least twenty-four hours. The infectivity of the defibrinated blood is destroyed by heating to 55° C. for fifteen minutes; and the defibrinated blood frozen for twenty-four hours is still infective at the end of that time. They were able to pro-

duce the disease in monkeys by placing them in a cage with sick monkeys during at least the early period of the disease. It was found that the incubation period of measles contracted in this way was not more than eleven days nor less than five. In a fifth paper they report on the infectivity of the nasal and buccal excretions from human cases of the disease. They found that the nasal and buccal secretions collected within the first forty-eight hours after the appearance of the eruption were infective for monkeys inoculated subcutaneously with this material. In a sixth paper they report on the duration of the infectivity of the secretions. Their results suggest strongly a reduction, if not a total loss, of infectivity of the nasal and buccal secretions in human cases of measles with the approach of convalescence. They tested out the infectivity of the measles scales and found, in six experiments, that in no instance were the scales infective. These studies by Anderson and Goldberger on the mode of transmission and etiology of measles virus is our first definite knowledge based on laboratory experiments as to the susceptibility of the monkey to inoculation with blood from cases of measles. They prove conclusively the infectivity of the nasal and buccal secretions during at least the first forty-eight hours of the eruption, and their experiments all tend to support their opinion that the scales in measles are non-infective. While Anderson and Goldberger are conservative in drawing their conclusions as to the duration of the infectivity of the secretions, their results are in accord with clinical observations that cases of measles are not infective after convalescence, which fact seems now to be well established. The importance of this latter point from a public health standpoint can be readily appreciated, for it has been the custom where quarantine is required for measles that the quarantine be maintained for twenty-one days from the onset. Based on the experiments of Anderson and Goldberger, it would seem that this period of quarantine can be safely reduced to ten days from the onset, as convalescence is usually well established in uncomplicated cases by that time.

PATHOLOGY AND BACTERIOLOGY

UNDER THE CHARGE OF

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Experimental Teratoid Tumors.—Two communications of much interest relative to the experimental causation of teratoid tumors were made at the International Pathological Congress at Turin. BORST (*Centralbl. f. allg. Path. u. path. Anat.*, January 15, 1912, xxiii, No. 1) reported 74 per cent. of successful inoculations of embryo emulsion, intraperitoneally, in white rats. Females were more readily inoculated

than males, although age was of little importance; the gravid state assisted the development of the tumors. Some "growth-stimulating" substances such as ether and indol used upon the emulsion, and in some cases injected after the tumors had begun to grow, proved ineffective in Borst's hands. Subcutaneous inoculation was unsuccessful, and animals in which this had been tried could be successfully inoculated afterward in the peritoneum. In addition to the tissues usually seen in such tumors the author has found brain-substance, lung, and spleen tissue. There was evidence in favor of autogenous formation of vessels, and of junction of such vessels with those of the test animal. ASKANAZY (*Ibid.*), who has priority in this work, reported further observations on the effects of "growth-stimulating" substances, used upon the embryo emulsion and also upon the test animal, and admitted that the results had scarcely come up to expectation. After subjecting the emulsion to ether solution and vapor for a day, 2 animals out of 8 injected subcutaneously showed growths of osteosarcomatous nature, both animals dying in the same length of time, these 2 being the only cases of malignant tumors seen in hundreds of animals observed. It certainly might be deduced that a heightening of the potency of growth of the cells had been obtained. The *x*-rays have a restraining action upon the potency; alcohol and lipase gave marked increase of growth, as did chloral hydrate, probably by inducing a cell-narcosis in the host-tissues. One animal so treated developed a large epithelioma. The presence of cartilage bone, glia, etc., speaks for the teratogenous nature of such a carcinomatous growth, but the question is still to be answered whether the malignant growth is essentially from the inoculated tumor or is a reaction on the part of the host-tissues. The extirpation of various organs has been practised and its effect upon teratoid growth observed; castration was of no effect; when the spleen was extirpated at the time or before the inoculation of 9 animals, 6 gave no tumor formation and 3 others but an insignificant degree.

The Bacterial Cause of Typhus Exanthematicus.—FUERTH (*Zeit. f. Hyg. u. Infekt.*, 1912, lxx, Part III) describes his research for the causative agent of "spotted fever," which he has observed in a considerable epidemic in Tsingtau, in the spring of 1911. Most of the cases occurred in Chinese coolies, but a considerable number of Europeans were also attacked, with a death rate considerably higher than in the case of the former. In 38 per cent. of the cases observed, a diplococcoid bacillus was found in the blood or the organs, which there is reason for supposing the causative agent. It is prone to exhibit marked variations when grown upon different media, in some of which, indeed, it appears as if it were a member of the streptococcus group, although its staining with Gram, its growth in milk and its morphological appearance are against this. Late in the disease, preferably four to two days before the fall of the fever, the blood culture is most likely to succeed, and at this time even smears of the blood may be successful in demonstrating the organism. The agglutination test proved positive only in low dilutions and in a few cases, and inoculation of pure cultures in apes was succeeded by a short febrile period after a considerable time of incubation. Once more the question is whether we deal with the actual agent or with a secondary infection frequently present.

The Disinfectant Power of Alcohol.—Beyer (*Zeit. f. Hyg. u. Infekt.*, 1911, Band lxx, Heft 2) details a most painstaking study of the effect of alcohol in various dilutions in killing pathogenic and pyogenic organisms. Seventy per cent. (by weight) alcohol far excels all other solutions in bactericidal power, being 30 times as effective as 60 per cent., and 40 times as effective as 80 per cent. alcohol. Beyer states that solutions under 60 and over 80 per cent. are useless for disinfection for practical purposes. Alcoholic solutions changing slightly by evaporation change greatly in their effectiveness. Absolute alcohol will not kill dried bacteria, and mixtures of alcohol with chloroform, ether, benzol, glycerin, etc., have not increased power over that possessed by the mixture with water. *Eau de cologne* is stronger bactericidally than the same strength of watery solution. Ethereal oils alone or in alcoholic solution are not practically useful, and carbolic acid, lysol, and kresol do not gain by solution in alcohol. Iodine in alcohol surpasses all solutions in power, and kills anthrax spores in one minute; even iodine in one-quarter per cent. solution is effective as a practical disinfectant. Decolorized iodine is weaker than colored, but is still very potent. Chlormetacresol and "eusapyl" gave excellent results as hand disinfectants, the alcoholic solution of the former ranking next to the tincture of iodine in its efficiency.

Appendicitis Caused by Parasites.—It will be necessary for most writers to revise their ideas upon this subject, if much more evidence be adduced similar to that brought forward by R. L. Cecil and K. Bulkley (*Jour. Exp. Med.*, March 1, 1912, xv, No. 3). Many will recall that Metchnikoff has for years pleaded the cause of the vermiform parasite in causing appendicitis, without making many converts. Cecil and Bulkley examined 148 appendices from children aged between two and fifteen years. Nineteen were normal, removed at autopsy, and of these 3 showed oxyuris; 129 were operative cases, and 19 of them showed parasites, 17 times oxyuris, and twice trichocephalus. In 15 out of the 19 cases, the parasites were associated with non-suppurative appendicitis, in 4 cases with a gangrenous form of the disease. The changes obsreved in the former (catarrhal) cases were confessedly slight, and Cecil and Bulkley state that in 70 per cent. of the 19 cases there were mucosal lesions unquestionably produced by the parasite. The oxyuris, they think, burrows into the lymph follicles causing extravasation of blood into the surrounding tissue, and often out through the mucosa. Hemorrhagic ulcers at times are seen; but it is stated that a characteristic feature of the oxyuris lesion is the absence of inflammatory reaction, which seems surely to be a strong piece of evidence against the authors' contentions in the matter. The trichocephalus tends to burrow beneath the epithelium. The conclusion which Cecil and Bulkley draw is that oxyuris and trichocephalus, when they occur in a diseased appendix, are, in most cases, the exciting cause of the pathological changes found. We may add that it seems almost necessary to know what is the state of the bowel in general with regard to parasites, before accepting fully the conclusions, for our own experience at the autopsy table indicates that most cases of parasites in the alimentary tract are in foreigners, that some of these are intensely infested with them; appendicitis in such cases would be

more likely to be accompanied by parasites than appendicitis in the native-born, common though oxyuris is in children of all nationalities. New York hospitals, too, are very certain to show, even among children, a good many foreign born. Some very beautiful photographs are shown of the parasites in the appendical tissue.

The Production of Antibodies by Artificially Grown Tissues.—It has been stated that "it is those cells in the body which anchor the toxins which produce the antitoxins," or otherwise, that the cell which is liable to attack is the one which forms the defense. A beautiful confirmation of this comes from A. CARREL and R. INGEBRIGHTSEN (*Jour. Exp. Med.*, March 1, 1912, xv, No. 3), who placed antigen with tissues which were cultivating artificially, and found that the newborn cells in the culture produced antibodies. Guinea-pig bone-marrow and lymph nodes were grown in guinea-pig plasma, and goat blood was used as antigen. Only cases whose technique was perfect, are reported, and it was found that the fluid of the cultures containing goat blood acquired a hemolytic power upon goat corpuscles, while the serum of control cultures remained negative in this regard. This hemolytic power was preceded by phagocytosis of the goat corpuscles by the guinea-pig leukocytes; no phagocytosis was seen in the first two days, but on the third day of incubation of the cultures phagocytosis was well marked. Hemolysis occurred without the addition of complement, by substances acting as natural hemolysins.

Means of Immunity in Bubonic Plague.—SIGNORELLI (*Lo Sperimentale*, February, 1912, Anno lxv, fasc. V-VI) details the trend of opinion voiced at the international conference at Mukden regarding means of vaccination and therapy in the bubonic plague. It is considered that while it is perfectly proved that vaccination is an efficacious means of defence against the bubonic form of the disease, it cannot be said that it is effective in the case of the pulmonary form of the disease. The best method, and the one most widely used, is that of the inoculation of dead bacilli (Haffkine), while the method of Lustig and Galeotti, of using the nucleoproteins of the bacilli, is also useful. Indeed, the production of antibodies agglutinative to the bacilli has been repeatedly confirmed, and is best furthered by the use, as antigen, of the nucleoprotein of the bacilli prepared according to the method of the last named experimenters. Serotherapy, it must be admitted, has proved quite ineffectual in the treatment of the pulmonary form of of the disease; the most that could be claimed was a prolongation of the course of the disease, and this only after the use of large quantities of serum.

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